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THE INFLUENCE OF THE KENNY CONCEPT OF ACUTE POLIOMYELITIS ON THE PHYSICAL TREATMENT THROUGHOUT ALL STAGES OF THE DISEASE *

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Those of us who care for the after-effects of poliomyelitis in all its stages are constantly faced with the question of whether the acceptance of the Kenny concept of the clinical manifestations in the acute stage of the disease necessitates drastic changes in all of our old routines throughout the entire period of the patient's rehabilitation.

It must be remembered that our previous concept of poliomyelitis was based almost entirely on clinical manifestations arising from an interruption of motor impulses to the affected muscles due to permanent or temporary damage to anterior horn cells in the spinal cord. We admitted that it was impossible to evaluate the damage accurately in the early stages of the disease. Our treatment was based on the hope that permanent damage was minimal and in time sufficient muscle fibers would again receive nervous stimulation to permit functional motion. I need not review the various routines of treatment or the results from them.

Miss Kenny came forward with an entirely new interpretation of the clinical manifestations of the acute stage. This concept maintained that the dominant factor responsible for the crippling effects of poliomyelitis was not a flaccid paralysis due to destruction of lower motor neurons, but actually a hypertonicity and irritability of the involved muscles setting up a sequence of events leading to apparent loss of voluntary contraction in muscles opposing the muscle in "spasm." This concept pointed out that contractural deformities of involved segments result from the sustained pull or continued hyperirritability of these muscles and not from the tonic pull of a normal muscle against a paralyzed one. Thus, the syndrome characterized by muscle "spasm," "alienation" and "incoordination" (to use Miss Kenny's own terms), which if untreated goes on to widespread contractures and apparent paralysis, is, in effect, an entirely new disease entity — new even to the extent that it probably arises from a lesion or lesions distinctly apart from damage to anterior horn cells.

While Miss Kenny at first vigorously denied the existence of a flaccid paralysis, the latest book on the Kenny concept adds "muscle weakness and paralysis" to the original triad of "muscle spasm," "incoordination of muscle action" and "mental alienation of muscle." Because of this, it must be stressed that the present concept does not deny the existence of the flaccid type of paralysis on which the whole framework of our former methods of treatment were built.

It is my opinion, based on the response of patients in the acute stage to early treatment consistent with the Kenny concept, that permanent interruption of all motor impulses to a bodily segment due to massive destruction of all of the anterior horn cells supplying that segment is a relatively uncommon com-

* Read before the combined meeting of the Boston Orthopedic Club and the New England Society of Physical Medicine, Boston, Mass., April 26, 1943.

plication of poliomyelitis. It seems apparent that untreated muscle "spasm," "alienation" and "incoordination" occurring in the acute stage are the dominant factors responsible for the widespread crippling seen in the later stages.

We must not confuse this Kenny concept of poliomyelitis with the Kenny method of treatment of that disease. The Kenny method is a logical, intelligent attempt to treat this new syndrome and prevent its crippling sequelae. As such, it is amazingly efficient, and we have no reason to believe that it has reached its peak of perfection. It is still in a state of evolution, and there is little doubt that in the future many of the present routines will be discarded and replaced by new and more efficient routines based on additional clinical experience coupled with the findings of basic research.

In the acute stage of the disease it should be obvious that we must accept the Kenny method in whole or in part, or, if we cannot accept the Kenny method, devise a new method of our own, because in this stage we are faced with a syndrome that we have never treated before. Experience has shown conclusively that patients in the acute stage do respond to early treatment directed against the manifestations of "spasm," "alienation" and "incoordination."

In the convalescent stage, the concept affects the reason for treatment more than it does the specific routine of treatment, and it is to this stage that we must give a great deal of thought. While this thought must be unbiased by our previous teaching and experience when we outline treatment for the manifestations of persistent muscle shortening, "alienation" and "incoordination," it must be tempered by our previous teaching and experience when we face manifestations of beginning muscle contracture and of true motor cell damage. In the chronic stage the concept may explain many of the results but offers little to change residual paralysis and deformity.

It thus becomes apparent that, while the basic concept of the disease remains unaltered throughout the three great stages, the primary treatment problem encountered in each stage may be quite different and may require a widely diversified physical approach.

In the acute stage the outstanding clinical findings are (1) persistent shortening, hypertonicity and hyperirritability of various muscle groups, usually coupled with muscle tenderness and pain; (2) marked muscle incoordination, and (3) loss or diminution of voluntary muscle power. The primary treatment problem in this stage is to make the patient comfortable and to release as quickly as possible persistent muscle shortening and hyperirritability. We are not primarily concerned with the loss of voluntary muscle power, because it is only after complete relief of pain and persistent muscle shortening that we can attempt to evaluate the cause of this loss. We cannot tell at this time whether it is due to "alienation," to destruction of anterior horn cells or to a combination of these factors. We may, however, be suspicious of severe damage to the motor units if we find either a diffusely weak segment showing little spasm and pain or antagonistic muscle groups in a segment showing profound spasm and pain. On the other hand, we may assume that "alienation" is the probable cause if the apparently paralyzed muscle is opposed by a persistently shortened and painful group.

All of us will agree, I am sure, that any record indicating the functional strength of individual muscles cannot be accurate in this stage; however, as a means of evaluating the patient's response to treatment, some type of permanent record of the clinical findings in the acute stage should be used. This record should list the muscles showing apparent paralysis and weakness and those showing spasm and incoordination. We have made up a special chart on which to record our findings in this stage. In all probability it will be changed many times in the future as our experience grows and indicates that changes are neces-

sary. This chart lists the important muscles, the so-called prime movers in most instances, and gives space to list the muscles in spasm and the degree to which the segment can be moved before lengthening of the involved muscle elicits pain or hyperirritable resistance. It also gives space to indicate whether voluntary motion is present, but we do not attempt to make a muscle test in the old sense. We simply put on this chart a zero if no motion is seen, one plus mark if weak, nonfunctional movement is present and two plus marks if motion appears to be of functional value. We also have space to indicate any gross incoordination. The chart can be filled out during the initial clinical examination and requires no additional handling of the patient. It is important to realize that the initial picture may change markedly from day to day during the first seven to ten days of the acute stage, so that a new determination of the position and extent of spasm should be made each day and recorded if significant changes have taken place. Figure 1 shows a portion of the chart.

Left	Right	Limitation			Inco-ordination	Limitation			Inco-ordination
		Passive Muscle Range	Type	Active Motion		Passive Muscle Range	Type	Active Motion	
		Date 3/1/43				Date 3/14/43			
Shoulder									
ELEVATOR—Upper trapezius		.....	S	++		.....	S	G	
DEPRESSOR—Latissimus dorsi		.....	—	+		.....	—	F	
FLEXORS—Coraco-brachialis		.....	—	O	Pect. M	.....	—	Tr	Pect.
Anterior deltoid		.....	—	O	Biceps	.....	—	Tr	Pect.
EXTENSORS—Teres major		.....	S	+		.....	S	P	
Posterior deltoid		.....	—	O		.....	—	P	
ABDUCTOR—Deltoid		.....	—	O	Pect. M	.....	—	Tr	Middle
ADDUCTOR—Pectoralis major		70°	S	+		85°	S	F+	
EXT. ROTATOR — Infraspinatus		.....	—	+		.....	—	F	
INT. ROTATOR—Subscapularis		.....	....	....		.....	....	....	
SCAPULA. ADD.—Rhomboids		.....	—	O	Middle	.....	—	P	
Trapezius		.....	—	O	Lower	.....	—	P	
SCAP. FOR. ROT.— Serratus anterior		.....	....	....		.....	....	....	
Elbow									
FLEXORS—Biceps		45°	S	+		10°	S	F	
Brachioradialis		.....	S	++		.....	S	G	
EXTENSOR—Triceps		.....	—	O		.....	—	P	

Fig. 1. — Portion of "Muscle Chart" used in acute stage of Poliomyelitis.

Our treatment in this phase follows strictly the principles laid down by Miss Kenny. We have attempted to keep abreast of the work done by the Minneapolis group in this phase and have tried to follow their routines as closely as possible. From our experience and from the work we have observed in

Minneapolis, we see no reason to challenge the claims made by Miss Kenny. Her results have been excellent, and time alone will tell whether they are permanent.

After muscle hyperirritability and acute pain subside we believe the patient to be in the so-called convalescent period. However, regardless of the time elapsed from the onset of acute symptoms, the patient is still considered to be in the acute stage if gentle passive lengthening of the involved muscle elicits jerky, irregular and painful resistance. Patients in the convalescent stage can be divided into two distinct groups.

The first group comprises patients with involved muscles that can be painlessly and passively brought to their normal extended length. There may remain obvious hypertonicity of certain groups, but there is no hyperirritability or jerky, involuntary resistance when the muscles are gently placed in their normal extended length.

Any combination of hypertonicity or flaccidity, voluntary power or apparent paralysis may be found in the involved segments, but no persistent shortening of muscle remains that cannot be overcome by gentle, painless passive motion. The patients in this group have usually had intelligent handling during the acute stage. They may or may not have had hot packs, but at least there has been nothing done to aggravate the so-called "spasm," and unsupervised, haphazard movement of muscles still under voluntary control has not been permitted. The treatment is clear cut. Hot packs may be applied to all segments showing hypertonicity or flaccidity. In my own experience, the application of hot packs appears to be of limited usefulness in this stage. Certainly so-called "two hour packs," that is, packs applied at two hour intervals five or six times a day, seem almost without value. Far better response is obtained from intensely hot, well wrung-out packs applied six to eight times at five minute intervals, with the procedure repeated several times each day. This repeated short interval packing is intended to act as a strong peripheral stimulus initiating proprioceptive impulses rather than as a means of simply applying heat to an involved extremity. Painless passive movement through the normal joint range is given to all involved segments several times daily. Muscle stimulation is given all muscles not under voluntary control, and active assistive motion is initiated in all prime movers under voluntary coordinated control. Weight bearing is initiated as soon as involved groups are free of pain, hypertonicity and incoordination. At Warm Springs we are utilizing lightweight corrective apparatus in the presence of incipient deformity and marked instability. For example, a walking splint is applied if there is any tendency to a genu recurvatum from relaxation of the posterior capsular ligaments of the knee. This splint is light in weight and allows free flexion of the knee but prevents further extension after a 5 degree flexion angle is reached. There is no binding of the joint, and the patient becomes accustomed to walking with the knee pointing forward and slightly bent. We may also utilize a light corset for short periods when the patient is first allowed to stand erect and walk. We do not feel that we are compromising the basic principles of the Kenny concept in any way by adding certain orthopedic appliances designed to assist the patient to carry out normal coordinated movement.

Response to treatment is usually dramatic. If little or no motor cell death has occurred, the segment is rapidly brought under efficient coordinated control (fig. 2). If gross motor cell destruction has taken place, there is, of course, but little return of power. Either the segment is completely flaccid or a diffuse return of voluntary contraction below functional capacity is seen (fig. 3). When voluntary power returns it is diffuse and well balanced. It is with unceasing

Name					Right Arm	
			10/29/41			5/16/43
Anterior	Deltoid	Anterior	O	Tr	P+	N
Middle	Deltoid	Middle	Tr	P—	P+	G
Posterior	Deltoid	Posterior	P—	P—	P+	N
Upper	Trapezius	Upper	P	P?	P+	N
Middle	Trapezius	Middle	P	P?	P+	G+
Lower	Trapezius	Lower	P	P?	Tr	N
	Serratus magnus		—	—	—	N
	Rhomboids		—	P+	F+	G+
	Latissimus dorsi		P?	F	G	N
Clavicular	Pectoralis Major	Clavicular	F—	G	G	N
Sternal	Pectoralis Major	Sternal	F—	G	N	N
	Outward rotators					

Fig. 2. — Chart of patient showing diffuse return of muscle power in right shoulder girdle over a seven months period of treatment.

Internal oblique		Internal oblique		P	P+	F+
	Transversalis			....	.....	.....
	Gluteus maximus			O	P+	F
	Ilio psoas			O	P	P+
	Sartorius			P	P+	P+
	Tensor fasciae latae			O	P	P+
	Hip abductors			Tr	P+	P+
	Hip adductors			P	P	P+
	Inward rotators			O	P	P+
	Outward rotators			O	P+	F
	Quadriceps		SEE	F	P+	F+
Inner	Hamstrings	Inner	WHITE	O	P+	P+
Outer	Hamstrings	Outer	SHEET	O	P—	P+
	Gastrocnemius		TEST	O	Tr	P
	Anterior tibial			F—	G—	G—
	Posterior tibial			P+	F+	F+
	Peroneals			O	P	P+
	Extensor longus digitorum			O	Tr	P+
	Extensor brevis digitorum			O	P	F
	Extensor proprius hallucis			O	P+	F
	Flexor longus digitorum			O	Tr	P
	Flexor brevis digitorum			O	Tr	P+
	Flexor lumbricales			O	Tr	P+
	Flexor longus hallucis			O	Tr	P+
	Flexor brevis hallucis			O	Tr	P+

Fig. 3. — Chart of patient showing diffuse return of muscle power over a five months period of treatment indicating strength still below functional capacity.

amazement that I see patients with very little muscle power carry out effective functional activity without the limping and lurching formerly seen. This is due to efficient coordinated use of all remaining neuromuscular units, unhampered by contractures and structural malalinement. It is interesting to note that we rarely see a spotty return of power in patients receiving treatment consistent with the Kenny concept (fig. 4).

	Gluteus maximus		N
	Ilio psoas		P
	Sartorius		P
	Tensor fasciae latae		N
	Hip abductors		N
	Hip adductors		P
	Inward rotators		N—
	Outward rotators		N
	Quadriceps		P
Inner	Hamstrings	Inner	N—
Outer	Hamstrings	Outer	N—
	Gastrocnemius		N
	Anterior tibial		P
	Posterior tibial		F
	Peroneals		N
	Extensor longus digitorum		G
	Extensor brevis digitorum		N—
	Extensor proprius hallucis		G
	Flexor longus digitorum		G+
	Flexor brevis digitorum		N
	Flexor lumbricales		N
	Flexor longus hallucis		N
	Flexor brevis hallucis		N

Fig. 4. — Chart of patient on admission one year after onset of Poliomyelitis showing grossly irregular return of muscle power.

Patients in the second group encountered in the convalescent stage are quite different from the first, and far more difficult to care for. Even when admitted early in the course of their disease they present deformities — equinovarus, tibial torsions, knock knees, pelvic obliquities and scolioses, to mention a few. They are likely to be difficult to examine because of extreme tenderness and pain in both muscles and joints. They are grossly incoordinated and substitute action of the few muscles still under voluntary control in all motions they perform. They are usually emaciated and discouraged. It becomes quickly evident that underlying the pain and limitation of motion are beginning fibrous contractures of muscle and of periarticular structures. Many of these patients have had hot packs, some of them given in the best possible manner.

It is becoming increasingly evident that it is as easy for contractures to develop in hot packs as in a plaster cast, regardless of how efficiently the packs are applied, if nothing is done to initiate early motion in the involved segments. The great value of hot packs and of meticulous nursing in the acute stage is to relieve pain and hyperirritability and thus minimize alienation and permanent

shortening by allowing early motion of involved segments. Our primary problem in the treatment of this group is to determine as quickly as possible the relative proportion of spasm versus contracture responsible for the limitation of motion. Any residual spasm must then be released as quickly as possible not only to prevent further contracture but to allow us to institute more vigorous therapy to release the existing contractures.

Every patient in this group deserves a trial of intensive hot packing coupled with intelligent routines intended to reestablish mental awareness and coordination, but if there is not appreciable change in the persistent shortening in four to six weeks more vigorous treatment must be instituted without delay. This includes active stretching on the table or in warm water, local heat and massage, even plaster wedging and weight bearing with suitable apparatus. It may be argued that such patients would never be seen if they were correctly treated from the onset — which may be true under ideal conditions of treatment — but we all know that we are still seeing patients in this condition, and it has been our experience that we cannot temporize with contractures of muscle and joint and with faulty structural alinement. We shall continue to see patients in this condition until it is realized that the mere application of hot packs is not a magic formula; and we shall continue to see such patients until their care is supervised by physicians trained to recognize and care for incipient deformity. All too frequently we see patients who have been treated month after month by hot packs and rest in bed. They are brought in to us with severe deformities, but only after their physicians sense that something must have gone wrong with the "hot pack treatment." They solemnly tell us that the "Kenny method" isn't much good in the bad cases. They usually have no idea whatever of the true Kenny method.

Following the suggestion of Dr. Herman Kabat, we have found that prostigmine appears to have a definite place in the treatment of convalescent poliomyelitis. We have not yet had the opportunity of using it in the acute stage but certainly intend to when the occasion permits. When a patient in the convalescent stage with persistent muscle shortening is admitted, a so-called prostigmine test is given. Prostigmine methylsulfate, 1.5 mg., is given subcutaneously. The joint range permitted by the shortened muscle groups is measured prior to the injection and again forty-five to sixty minutes afterward. We feel that this test may be of value in determining the extent of underlying contracture responsible for the persistent shortening and thus indicate certain routines of treatment. If the increase in joint range exceeds 10 degrees we feel that the patient should benefit from continued daily use of the drug. This is given orally in the form of prostigmine bromide, 30 mg. three times a day. Atropine sulfate, 1/100 grain, is given with each dose of prostigmine. We also feel that prostigmine has a decided beneficial effect on coordination and should be used when the patient shows evidence of incoordination even though a prostigmine test fails to demonstrate any appreciable lengthening of involved muscle groups. In fact, the effect on coordination appears to be far more consistent than the effect on persistent shortening of muscle. These observations, however, are based on limited experience, and we realize that changes in the degree of incoordination are difficult to evaluate accurately. We have had no ill effects from the administration of prostigmine on any patient so far and have given it in relative doses to infants and children.

In the chronic group we place those patients who in the course of time and conservative treatment have reached a stage where no further increase in their functional capacity can be expected. The Kenny concept can help explain the results, and perhaps point out the deficiencies of past treatment, but it offers

nothing to correct the situation at this late date. Our primary problem is to evaluate the patient's disability in terms of possible worth while activity when he returns to his own home. It is evident that even though a patient can stand and even walk without the aid of orthopedic appliances it may be impractical and even dangerous for him to do so. The application of indicated supportive apparatus, with or without surgical stabilizations of affected joints, may be necessary to secure stability and to permit the patient safely to earn his own living or maintain his own home. The management of the deformed segment may require repeated surgical corrections until optimum alinement and balance of remaining muscle power is obtained. The Kenny conception aids this problem in no way.

Conclusion

While the full acceptance of the Kenny concept is hampered by poor terminology and by our limited knowledge of many of the basic physiologic and pathologic facts responsible for the cause and the after-effects of poliomyelitis, it is proving itself clinically sound. In the acute stage of the disease this concept presents a syndrome of clinical manifestations which may lead to widespread weakness and deformity unless immediately recognized and treated. No one of these clinical manifestations is new in the sense that it had never been seen before, but the full significance of the triad making up the syndrome had not previously been appreciated. It is evident that, since we had not been aware of the full significance of these clinical findings, our past forms of treatment are likely to be inadequate and must either be discarded or be modified in ways consistent with the new concept. We must not confuse the Kenny concept with the Kenny method. The Kenny concept is a presentation of clinical findings in the acute stage of the disease. The Kenny method is an attempt to treat these findings.

In the convalescent stage the concept explains the cause of the development of contractural deformities and the spotty return of muscle power, and in this way indicates certain routines of treatment. These routines may be similar to those used in the past, but the reasons for their use may be quite different. In this stage there must be no temporizing in the face of beginning periarticular and intramuscular contractures. We have in the past learned how to deal with these contractures. We must not sit idle and hope that hot packs will dissolve the contractures in some magical manner. If they do not quickly respond to conservative measures they must be stretched out manually or even wedged out in plaster casts. We have long known the danger of allowing patients to bear weight and increase their activity unless correct structural alinement can be maintained. When the patient's activity should be increased and protective or supportive apparatus is indicated, such apparatus should be utilized as long as is necessary. Our statistical results may suffer but our patients won't.

In the chronic phase of the disease the concept helps explain the results and perhaps points out the inadequacies of past treatment, but it solves the treatment problem in no way.

ANALYSIS OF TREATMENT OF INFANTILE PARALYSIS *

With Comments On Kenny System

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Much publicity has been given in the past year or two to the Kenny treatment for infantile paralysis in its acute stage. As a result, there has been considerable controversy as to the relative merits of this system versus the former method of treatment, which Miss Kenny refers to as the "orthodox" method. In an evaluation of the relative merits of the two, a brief review of the previous practice and some details of the Kenny system will be presented.

When Miss Kenny speaks of the "orthodox" treatment, she probably refers to the type of treatment given by physicians who had little experience or training in the care of patients with poliomyelitis in its acute stage. She may refer to patients who had plaster applied in the early days of the illness and wore it uninterruptedly for some weeks or months. Then, they may or may not have been fitted with braces and either were not treated by muscular reeducation or other physical therapeutic measures or received treatment which was grossly inadequate when judged by any recognized standard. In the chronic stage of the illness surgical intervention was carried out to correct deformity which might have been prevented or to rehabilitate the patient.

If this is Miss Kenny's concept of the "orthodox" treatment, I am in complete accord with her condemnation of the method. However, such treatment differs materially from that given patients under the guidance of such workers as Bennett and Kendrick at the Children's Hospital School in Baltimore; Ober and Miss Merrill at the Children's Hospital in Boston; Lowman in Los Angeles; Hoke, Irwin and Miss Plastridge in Warm Springs, and the workers at the Betty Bacharach Home in Atlantic City, N. J., to mention a few.

Because of the publicity given the Kenny system in "yellow journals" and other lay publications, many laymen and even physicians who have not thoroughly studied the subject of infantile paralysis are of the opinion that the Kenny system consists of the application of hot foments and that the patients, with perhaps rare exceptions, make complete, rapid and uneventful recoveries. Miss Kenny's concept of the "orthodox" treatment differs as much from the "previous" methods used at recognized centers, such as those mentioned, as the application of hot wet blankets differs from the Kenny system.

In the past, most patients with infantile paralysis were not seen for active treatment by the orthopedic surgeon until they had reached the chronic stage of the illness. The orthopedic surgeon, as well as other physicians, usually did not have the opportunity to see, study and treat any sizable group of these patients. A resident at a large New York orthopedic hospital told me that in a four year period he had seen only 3 patients with infantile paralysis in the acute stage. Most general hospitals, and even orthopedic institutions, because they are interested in a relatively rapid turn-

* From the orthopedic service of the author at the Betty Bacharach Home.
* Read before The Pennsylvania Academy of Physical Medicine, March 18, 1943.

over, cannot be expected to treat patients for a period of two years or longer, the duration, not infrequently, of the acute phase of infantile paralysis. Thus, except in a special institution to which such patients are referred from a relatively large area, the staff may not see or treat enough patients to be adequately trained in the work. It is not surprising, therefore, that in recent discussions of this subject, both in the literature and at various local and national meetings, the essayists and discussors for the most part have acknowledged that their remarks were based on a study of the literature and their recent experiences. Their ideas and thoughts, therefore, had to be based on impressions.

At the Betty Bacharach Home in Atlantic City, about 500 patients with infantile paralysis in the acute stage were studied between 1924 and 1942. These patients were referred from all parts of New Jersey as well as from eastern Pennsylvania and New York. The Betty Bacharach Home is staffed with workers, nurses, occupational therapeutists and physical therapeutists who have been specially trained to care for patients in the acute stage of infantile paralysis. The patients are admitted soon after discharge from contagious disease hospitals, and treatment is continued for the most part as long as the general and muscular condition continues to improve—not infrequently for a year and sometimes for two years or even longer.

In the years 1939 to 1942, a group of 147 patients were treated by the so-called "orthodox" method. Detailed statistical records are available for this group. At the present time, the patients at the Betty Bacharach Home are being treated by the Kenny system, and the results obtained will be compared with those obtained in treating the 1939-1942 group of 147 patients. This comparison will be of greater value than previous statements, based on impressions, even though differences in muscle involvement, susceptibility, virulence and other factors are acknowledged.

The acute phase of infantile paralysis may be defined as the stage beginning with the onset of the illness and lasting until maximum muscle power has been regained with the help of continued active treatment. Or, it may be defined as the period between the onset of the disease and the chronic stage.

After a twenty year study of patients in the acute stage of infantile paralysis, it became possible to make a fairly accurate prognosis within the first few days or weeks of the illness as to the relative recovery of muscle power. The general ultimate recovery, rather than the rapidity of recovery, could be surmised. The prognosis was possible even though detailed muscle testing was not done. The prognosis would be considered favorable if some degree of muscle power was present in most of the muscle groups of an involved extremity. At times, even though no actual muscle power could be observed, the prognosis was considered guardedly favorable if the part had a relatively normal topography of the respective muscle bellies or if the attitude of the part and the general muscle tone were relatively good.

For twenty years students have been taught at Jefferson Medical School that patients with infantile paralysis may have complete recovery, with no residual muscular weakness or paralysis, or may have some degree of recovery. If the anterior horn cells have been destroyed, the muscles affected will be permanently paralyzed. However, if the anterior horn cells are only partially or temporarily damaged, the muscles may recover or be left weaker than normal, depending on the ultimate degree of damage to the nerve cells. The early apparent lack of power in a muscle may be due to toxicity associated with the illness or to a phase of memory loss for the use of the muscle, a symptom which Miss Kenny calls "mental alienation." The students

were taught that with proper treatment patients with this involvement might recover.

In our work we have observed a group of symptoms which we have called "general nervous system reaction." It is an early symptom of the illness and may continue for months, a year or longer. The patients are often jittery, apprehensive and irritable and cry readily, or they may be listless and unable to cooperate; also they may be overanxious, tire easily and have poor resistance. Abatement of the general nervous system reaction is discernible in the patient's appearance of good health. Muscle function is more efficient and requires less effort. The patient is peaceful, and coordination and cooperation are good.

"Previous Treatment" of Infantile Paralysis in Its Acute Stage Used at the Betty Bacharach Home

Patients would frequently be admitted directly after discharge from the contagious disease hospital. They might be brought by automobile, ambulance or train a distance of 50 miles, more or less. Formerly, they were not splinted during their stay in the contagious disease hospital. More recently, however, through the good offices of Mr. Joseph Buch, chairman of the Crippled Children Commission, and at the suggestion of the medical director and orthopedic surgeon of the Betty Bacharach Home, the cooperation of workers at the various contagious disease hospitals throughout New Jersey has been enlisted in having the patients splinted immediately on admission to these hospitals and until they arrive at our institution. The children who were not splinted were usually jittery (general nervous system reaction), more exhausted and apathetic or restless, irritable and hypersensitive. Those who had been splinted were in better condition.

On admission, the patients were placed on Bradford frames. Since 88 per cent had weakness of the abdominal muscles a scultetus binder was put about the abdomen for protection. If extremity weakness was present, Toronto splints were used. The haggard, worn look often seen on admission soon disappeared, and the children had a more quiet, soothed and relaxed appearance.

Back pain and stiffness were frequent. Spasm of the back muscles was so severe at times as to cause the trunk to assume an attitude simulating opisthotonus. To prevent further irritation of these muscles, the patient was placed on a convex stretcher frame, and, as the spasm abated, the curve in the frame was gradually straightened. The application of heat for a period of one hour with a 1,000 watt bulb was found to be both relaxing and comforting and was repeated several times daily. If the precursory examination made after the child had reacted showed weakness of the abdominal muscles, a Hoke corset was worn until maximum recovery had occurred.

If there was weakness in the upper extremities, Toronto splints were soon replaced by accurately fitted airplane splints. For involvement of the lower extremities, circular plasters were applied as soon as the patient's general condition warranted. For weakness below the knee, the foot was placed in an attitude to relax the weaker muscles. The knee was held in slight flexion, and a board parallel with the bed was incorporated in the plaster to hold the extremity in the degree of rotation necessary to relax the weaker group of rotator muscles. The plasters were bivalved when they were dry. Stockinet covered the inner surface of the plaster, and the outer was protected with a coating of celluloid in acetone, by white lacquer

or by wood varnish. Straps and buckles were attached with staples, so that the splints could be readily applied and removed.

Muscle pain and tenderness and joint stiffness were frequent early signs and were treated with heat but with minimum handling of the patient. As previously stated, heat with a 1,000 watt bulb was used to help relieve back pain, spasm and stiffness, and it was used, too, for muscle tenderness in other parts. As soon as the patient's condition permitted and while muscle tenderness was still present, the patient was wheeled in bed to the Hubbard tank for hydrotherapy. In order to minimize handling, the patient was lifted from the bed into the tank with the aid of a hoist. At all times the extremities and trunk were maintained in an attitude to protect them from undue strain. The patient received a warm soap and water shower before entering the Hubbard tank or pool, both of which contained salt water pumped from the inlet immediately adjacent to the institution. The water was heated to a temperature of 98 to 102 F. for the Hubbard tank and 94 to 96 F. for the pool. The effects of the moist heat were comforting and relaxing and helped to relieve the tenderness and joint stiffness.

Whirlpool was a further aid in correcting joint stiffness. A powerful motor was fastened to the lower end of the tank at each side to create the whirlpool effect. Since one patient was treated in the tank, he could be handled with greater ease, there was less distraction and it was ideal for the patient who was frightened or apprenhensive. As discomfort abated and the patient's general and local condition improved, treatment was given in the pool.

After hydrotherapy, the patient was dried and the parts again supported in a proper attitude until the splints were applied. Gentle massage was also used, not so stimulating as to increase pain or so sedative as to further weaken the parts, but sufficiently gentle to be comforting.

Almost from the beginning, the joints were moved through their painless range of motion. Muscular reeducation was started as soon as the patient's condition warranted and was repeated at least twice daily, once in the tank or pool and again, later in the day, either in bed or on the treatment table.

Recumbency was continued until the general nervous system reaction had abated and the patient's general condition had improved and as long as muscle power continued to improve. When the abdominal muscles and the gluteal extensors had a rating of 80 per cent, the patient was permitted to sit up for short periods. As his resistance improved, the time was gradually increased, but care to avoid fatigue was a constant aim. However, if after nine to twelve months of recumbency, the weakness of the abdominal and extensor thigh muscle persisted, the patient was permitted to sit up for gradually increasing periods. This practice rarely decreased muscle strength, and not infrequently a spurt in muscle power was observed, as well as an improvement in the general condition. Later the patient was permitted to sit out of bed, at first for fifteen minutes daily and then longer, until he was out of bed most of the day in a wheel chair. He was not permitted to bend his legs unless there was sufficient muscle power or the parts were protected in splints or braces.

As the general strength improved and the muscle power increased to a maximum with the patient recumbent, sitting and then standing were allowed. If normal muscle power was present except in one or two musules, standing was allowed. Standing and walking in the pool were started as soon as the muscle picture warranted and joint stiffness and the general nervous system reaction had abated. Then the patient was permitted to

stand for a few minutes at the conclusion of a muscle exercise period; as improvement progressed, the time was increased, with particular attention to preventing fatigue.

If muscle power in the lower extremities was normal, walking was permitted without support, or, according to the muscle paralysis, walking was permitted with the aid of ankle or leg braces. Canadian crutches were used until the patient learned balance and symmetry of gait, and walking with a four point gait was the aim. The patient was always fitted with sturdy, well balanced oxfords when permitted to be ambulatory; bedroom slippers were forbidden.

While a muscle power of 50 per cent might be adequate for the patient to function without braces, support was not discarded while there was continued improvement in the muscle picture. When maximum power had been obtained by the treatment, changing the treatment sometimes resulted in a spurt in muscle power. As soon as greater use of the part was indicated. brace stops restricting the range of joint motion were opened for a few minutes, then gradually longer, each day. Changing a leg to an ankle brace or removing the hand or forearm part of an airplane splint was a later step in the treatment, but a power of 80 per cent or normal was desirable. When the strength of the part was 90 per cent, braces could be discarded entirely.

While the muscle power usually returned within the first six months, a gradual increase of power for a year or even two years or longer was not unusual. At times it was observed that muscle groups suddenly began to improve even after a year and continued to improve until sufficient power had been acquired for good use of the part. Stiffness in the back and joints of the extremities was often found to mask power that was present, so that a notation was made on the chart to the effect that increased power was to be expected when stiffness had abated. Subsequent experience showed this idea to be sound. Perhaps the most frequent example was the return of power to the anterior abdominal muscles when back stiffness was relieved. As a rule, power returned sooner in the oblique and transverse than in the anterior abdominal muscles.

The aim of treatment was to obtain balanced power in the muscles rather than to see how much power might be obtained in any one muscle or group, so that in muscular reeducation, primary attention was directed to the weaker muscle rather than to the stronger one.

Muscle reeducation was continued as long as testing showed that an increase in balance and power was continuing. The first detailed muscle testing for charting purposes was done when the patient's general condition permitted. Because of the general nervous system reaction and the effects of the illness on the body in general, it was often three months after the onset of the disease before an evaluation of the muscle picture could be accurately made. Muscle testing was done in the early stages at monthly intervals and later at intervals of six to eight weeks.

Occupational therapy was important in the reeducation of muscles. A child might work on a loom to strengthen a weakened upper extremity muscle, but if the muscle was unprotected and permitted to stretch unduly during the exercise, more harm than good might result.

The work at the Betty Bacharach Home was supervised by the orthopedic surgeon, but most of the treatments were given by the physical therapeutists assisted by nurses and male workers trained in the proper handling of patients with infantile paralysis. Our physical therapeutists were obtained, or I might say imported, from areas far and wide. One worker came from Warm Springs, another from Colorado, one from Ann Arbor,

one from New York and several from Boston. These workers were taught to apply plaster, to bivalve plaster and to make splints. They were instructed in the proper fitting and adjustment of Hoke corsets, airplane splints and leg and ankle braces. They were taught the proper fitting and balance of shoes. The nurses, workers and occupational and physical therapeutists made weekly rounds with the orthopedic surgeon, and at stated intervals the brace man was included on these rounds.

After twenty years of work with many patients having infantile paralysis in its acute stage, one's experience and ingenuity may still be taxed, on occasion, in ascertaining and analyzing balance or imbalance in a part, in determining the best method of treatment or in analyzing the activity of a patient in order to prevent incoordination and correct muscle substitution. This has always been an aim in the treatment of patients with infantile paralysis through the use of muscular reeducation.

The Kenny System

In discussing the Kenny system, I shall omit detailed definition and anatomic, physiologic and neurologic explanation of the terms used by Miss Kenny, because that phase of the subject has previously been presented.

For thirty years Miss Kenny has been interested in the treatment of patients with acute poliomyelitis. Through the process of trial and error she has evolved a treatment which she believes to be of benefit to patients in this stage. Her ideas are still changing. Because of her lack of training, her nomenclature often disregarded accepted terms. Also, because of lack of scientific training and temperament, she probably reasons: "I did this. I do not have to prove what I have shown in a practical way to be right." Furthermore, some of Miss Kenny's theories of muscle function differ from those usually accepted by anatomists. However, scientists associated with the National Foundation for Infantile Paralysis are trying to explain the Kenny system on a scientific basis.

A discussion of the Kenny system might well begin with the statement that in principle it differs but little from the treatment previously used in recognized centers throughout the United States that were staffed with workers trained in the care of patients with infantile paralysis in its acute stage. The method may differ in the application of the principle and even more in degree. The principles of treatment may be readily learned, but practice and technical perfection are necessary for their proper application, and the method suggests the need for mastering many details.

It is still to be proved that patients with infantile paralysis in the acute stage treated by the Kenny system have less residual paralysis or more rapid recovery than those treated by the previous method. However, it might be proper to expect that local treatment of the subjectively very annoying early symptoms might favorably influence the prognosis of the illness and that with efficient care the effects of the virus might result in minimal ultimate damage.

Monkeys may be infected with the virus of infantile paralysis instilled into the nostrils; it is possible to protect the animal by first severing the olefactory nerves. Might it be that the virus travels along the nerve sheath to reach the cord? Conversely, might it be possible that the virus travels along the nerve sheath from the cord to the periphery to cause the symptoms of joint and muscle tenderness?

Miss Kenny describes three symptoms and directs her treatment to their alleviation. These are muscle spasm, muscle incoordination and mental alienation. More recently, she has agreed to accept the fact that flaccid

paralysis does occur even in patients treated from the onset of the disease by her method.

The orthodox method recognized that flaccid paralysis occurred in about 20 per cent of patients owing to the effects of the virus on the anterior horn cells of the cord. Recently, Miss Kenny, too, has acknowledged paralysis similarly associated with the effects of the virus, but not until the patient has had the benefit of adequate treatment for a period up to two years. She is of the opinion that true paralysis constitutes no more than 10 per cent of the total. According to her concept, the other muscles do not function because of spasm, usually of the affected muscle. If a muscle is stretched by an opposing muscle which is in a state of spasm, it cannot contract until returned to its normal resting length. In her concept, the virus for the most part has not affected the anterior horn cells or sensory motor mechanism. It is her opinion that the muscles are not functioning temporarily because of pseudoparalysis due to a state of mental alienation.

Muscle spasm, as Miss Kenny speaks of it, is so closely concerned with the symptoms of muscle tenderness and joint stiffness decribed in the previous method as to permit them to be considered together for the purpose of discussion. Miss Kenny defines muscle spasm as a physiologic condition of skeletal muscle with exaggerated tonus tending to cause muscle shortening. The affected muscle is usually the spastic one. Spasm suggests local irritability, is associated with and may be the sole cause of pain and has a disrupting influence on the neuromuscular system.

According to previous teaching, infantile paralysis may be defined as an acute infectious systemic disease caused by a filterable virus which has a predilection for nerve tissue, especially the anterior horn cells of the cord, and may result in a flaccid paralysis. Since the Kenny concept for the most part is in agreement with the previous concept of the etiology and pathology of infantile paralysis, it may be stated that the disease is essentially in the cord and that treatment directed to the trunk and extremities cannot be expected to have more than a favorable influence on the cord, the site of the major pathologic changes.

The opponent muscle is apt to be flaccid because of mental alienation. Spasm may persist for six months or longer and may recur. Constant attention is necessary to detect and treat spasm. Miss Kenny states that spasm is more damaging to bodly mechanics than paralysis, that it is the primary cause of deformity and that joint stiffness is actually unrelaxed muscle spasm or the contractures associated or resulting from it. Her criteria for the diagnosis of spasm in the various muscles are worthy of study, and her treatment for spasm is worthy of real trial.

Incoordination results in muscle substitution, and the term has always been recognized. It is an unphysiologic condition that predisposes, permits or determines the substitution of one muscle for another which would normally perform a certain movement. Muscle substitution occurs to aid function when there is organic or functional weakness or paralysis and results in incoordination. It is the result of mental alienation in which the impulse for movement may be suppressed or diverted to other channels and results in abnormal patterns of motion. An ineffective contracture occurs instead of a normal, rhythmic, coordinated movement because the subconscious mind accepts the changes associated with mental alienation as permanent. Treatment in both methods is by muscular reeducation and suggests supervision for prevention and correction of incoordination. Training and selective muscle exercises are used to prevent muscle substitution. Even

after the motor sensory arc is reestablished, muscular reeducation may be necessary before coordinated muscle action again occurs.

Mental alienation differs in no way from what was referred to in the previous method as "memory loss for the use of a part." According to Miss Kenny's concept, mental alienation is the mental loss for function of the nerve arc with inability to produce a voluntary, purposeful movement in a muscle despite the fact that there is no involvement in the sensory motor mechanism and the anterior horn cells are not organically destroyed. The muscle becomes alienated from the voluntary center which regulates motion and may result from fear, pain, toxicity or the effects of the disease itself.

In mental alienation it is usually the unaffected opponent that is stretched beyond its normal resting length. Because it is overstretched, it cannot contract from its point of attachment. The failure of the muscle to function is on a physiologic basis. It is not a condition of paralysis or muscle weakness as it might be graded in a muscle-testing examination according to the "orthodox method." Miss Kenny states that the former treatment would be splinting, but she advocates the use of hot foments to correct the muscle spasm and, later, uses muscular reeducation. She reports that there are fewer deformities and better function and that bone growth is not retarded. She stresses the importance of treatment to restore the nonfunctioning, nonparalyzed muscle. Unless mental awareness is reestablished, the use of the temporarily paralyzed muscle may disappear from the subconscious mind.

The aim of treatment in both the previous and the Kenny system is protection and muscular reeducation. Both methods, in a sense, splint the parts. The orthodox method protects by the use of splints, not rigid, long-continued plaster immobilization, but rather splinting as described earlier in this paper. While Miss Kenny does not use splints in the accepted sense, nevertheless she expects patients, especially in protecting the parts that are weak, to remain in an attitude simulating the normal standing position. She recommends the use of a foot board but does not consider it a splint. Still, the feet may rest against it and be maintained in a right angle position, even though its acknowledged purpose is to maintain the normal standing, Stutz reflex, by stimulation through the proprioceptive reflex. The foot board also relieves the potential deforming influence of the weight of the bed covers on the feet. A pad or support is placed beneath the knee to prevent back knee, and a rolled blanket may serve to protect a very weak lower extremity from undue rotation. Might this not be described as physiologic or even psychic splinting, provided the patient can maintain the necessary attitude?

Most parents find it difficult to control or adequately direct the activity of healthy children, and they are apt to be more indulgent with a child who is ill. Can most parents adequately supervise the child treated at home or most workers supervise the child treated in an institution, so that he will maintain a proper attitude to rest the involved parts? The necessary degree of rest is emphatically stressed by Miss Kenny's suggestion that in the treatment of a weak muscle with muscular reeducation the technician should do the exercise twice passively and permit the patient to do it only once actively. It would seem that splinting in a physiologic attitude and in the manner previously used at the Betty Bacharach Home might give more rest to the part than the patient's effort to rest it without protection and support.

Heat is used in both methods. Sister Kenny advocates the use of heat

more intensively than it was used in the previous method for the symptoms of muscle pain and joint stiffness (muscle spasm). If study shows that muscle tenderness and joint stiffness are more readily prevented or corrected by her method, we will be greatly indebted to Sister Kenny for a valuable change in the technic of applying heat.

Miss Kenny suggests that hot foments be started immediately with the onset of the illness; also stimulation to the proprioceptive reflexes by stroking the tissues adjacent to the tendons and moving the joints through a small arc of motion. It is our suggestion that this be deferred until the temperature is normal and the patient has reacted from the signs and symptoms of the acute systemic phase, which is usually within a few days after the onset of the illness. It does not seem right to burden a patient who is very ill with the systemic phase of an acute disease; rather, one should add to his rest and comfort. Certainly, if respiratory spasm is present, its immediate treatment is imperative.

In the Kenny system, muscle testing, massage, splinting and the use of the respirator are not advised. We cannot agree with Miss Kenny's dictum against muscle testing. Miss Kenny is not interested in percentage grading, but in coordination with smooth, rhythmic functioning, without regard to muscle strength. She reasons that if a muscle has the ability to contract smoothly, then with treatment it can be made to perform adequately. She states that testing, since its aim is to ascertain maximum power, causes a state of mental alienation and that the result is incoordination. Certainly, as previously described and used in our work, muscle testing did no harm, and it does provide a unit of measurement of the patient's progress. Also, the records may provide a basis for comparing the results obtained by the orthodox and Kenny methods. Miss Kenny analyzes the patient's muscle picture on her daily rounds as a criterion for treatment. This does not differ too much from our own practice, except that when the patient's condition does permit and warrant, a detailed muscle test is made.

I have expressed my views on massage and splinting. As to the use of hot foments versus the use of the respirator for respiratory embarrassment, I have no argument with Miss Kenny that previously the patient with bulbar paralysis did poorly in the respirator, or that if respiratory embarrassment will not be helped by treatment with hot foments, the patient will probably not be saved by treatment in the respirator. If respiratory embarrassment will respond more readily to the use of hot foments, then by all means let us ascertain this fact and adopt hot foments in the treatment.

While the first consideration in the treatment by the Kenny system is the use of hot foments to overcome muscle spasm, muscular reducation is started almost simultaneously. This is done in the early days of the illness to create mental awareness by stimulating the proprioceptive reflexes by gently rubbing the tissues at both sides of the tendon, or by passive motion of a joint through its painless range. When a trace of power is present, the aim is to increase it by muscle stimulation.

Miss Kenny describes "stimulation" as one form of muscular reeducation and considers it a powerful factor in restoring function by overcoming mental alienation. She defines it as "stimulation through the receptor in the peripherae to the proprioceptor in the subcutaneous tissue from the point of attachment." Its use is suggested primarily for those muscles which only show a flicker of power. She has described a technic for its practice in the treatment of practically every muscle.

As spasm abates and the patient's condition improves, treatment is continued by muscular reeducation. At the Betty Bacharach Home the Legg-

Merrill system of muscle reeducation is used, and the Kenny method differs but little and is probably based, for the most part, on the same system. The aim is to obtain the maximum balanced power according to the damage to the anterior horn cells and to accomplish this with all muscle groups working in a rhythmic, coordinated manner. While in many patients this may be rapid, in others continued treatment by muscular reeducation may show a gradual increase and return of muscular power. We are in accord with Miss Kenny that treatment should be continued by palliative measures until all possible muscle power has returned. This not infrequently means continued active treatment for a year or two or longer.

Miss Kenny's criteria for permitting a patient to sit up, get out of bed, become more active and become ambulatory does not differ in general from previous criteria. She does not advocate the use of braces if it is at all possible for the patient to get along without such support. If a brace is necessary to assist the patient in walking, she desires that he use it only while walking, so as not to interfere with the normal range of motion. Furthermore, a brace might favor mental alienation and therefore incoordination and muscle substitution. The parts must be kept supple and long and muscle spasm prevented. It is generally agreed, however, even by the most ardent advocates of the Kenny system in this country, that when the chronic stage is reached the methods previously used are still applicable.

It may therefore be stated that when all possible power has returned, the aims of the Kenny system have been satisfied and the patient has reached the chronic stage, braces may be necessary to aid activity, permit maximum efficiency and minimize strain. Reconstructive surgical intervention may then be needed, those muscles which remain being utilized to carry on the function of the part in as nearly normal a manner as possible.

Comment

In considering the merits of the two systems of treating patients with infantile paralysis in the acute stage at the Betty Bacharach Home, I am reminded of my own early training in the treatment of fractures. My first preceptor in the care of fractures was Dr. T. Turner Thomas, who was essentially a one man fracture team. Dr. Thomas reduced the fracture and fixed the part in plaster until healing occurred. My further training in the fracture field was under the guidance of Dr. Joseph Blake and his coworkers, who were ardent advocates of traction and suspension. Their results were excellent, because the entire personnel was trained and the patient had twenty-four hour a day supervision. Correlating this with the treatment of infantile paralysis, it may be stated that the results of either method depend in a large measure on the aptitude and training of the workers.

At the Betty Bacharach Home, the workers, physical therapeutists, occupational therapeutists, nurses and nurses' helpers were carefully trained in the treatment of patients in the acute stage of infantile paralysis. Since the advent of the Kenny system, a guide was prepared for their direction, and the workers in charge have taken several courses in the Kenny system, both for their own knowledge in treating patients and for training the personnel in this so-called "newer" method. As Director of the work, I have been interested in the study of infantile paralysis for two decades and have studied not only the orthodox but the Kenny system.

Scientists, anatomists, pathologists and authorities on infantile paralysis are still studying, observing and evaluating the Kenny system. A comparison of the results obtained in our present cases, in which treatment is by the Kenny system, with those of the past may help provide an answer as

to the superiority of one method or perhaps as to the most valuable phases of each. As previously stated, there are relatively few workers who have treated any great number of patients with infantile paralysis over a long period, and I know of no school in this city which majors in training workers in muscular reeducation. It would seem that unless the patients are treated by competent, trained workers, the end results may be found to be more favorable by the former use of splinting than by the Kenny system. Time and the accumulation of statistical data are needed before a definite answer is possible. It is my personal impression that the use of intensive heat and more frequent manipulation of the joints through their painless range of motion may result in more rapid relief of muscle tenderness and an increase in the range of motion in the joints.

If heat, as Miss Kenny suggests its use, relieves tenderness, and if the normal range of motion in the joint occurs sooner, then a valuable addition to the treatment of infantile paralysis has been found. The orthopedic treatment for the acute phase of infantile paralysis, as it was previously practiced at the Betty Bacharach Home and in other such centers, gave very good results in a large percentage of cases. However, paralysis and deformity also occur in many. The answer to many questions in the Kenny system are as yet both incomplete and unsatisfactory, but many of the things Miss Kenny does are logical. Until a preventive or specific treatment has been found for infantile paralysis, some patients will recover completely and others will have more or less residual paralysis, regardless of the method of treatment or whether it is started immediately or soon after the onset of the illness.

It is my impression that the treatment of the acute phase of infantile paralysis, will, for the most part, revert to the previous methods used at the Betty Bacharach Home and will incorporate those measures suggested by Miss Kenny that have proved their value. In either system of treatment, however, the value can be measured only by the expertness and training of the people who give it.

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THE FUTURE OF REHABILITATION *

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WASHINGTON

When I accepted the invitation to speak to you I had reason to believe that by this time legislation providing for a strong and comprehensive rehabilitation program would have been passed. It was my intention, therefore, to outline to you our revised and expanded organization, and to discuss the ways and means by which you and we may work more closely together. Unfortunately, because the program involves so many groups, both government and private, it was not possible to obtain agreement on a bill until it was too late for the retiring Congress to act on it. I can report to you, however, that agreement has now been reached and legislation should be passed early in the new session.

I wish to talk about the future of rehabilitation from the standpoint, first, of present and future need for such a program and, second, of the obstacles that must be overcome if it is to function as an effective social instrument. I am going to talk about it because the time has come when we can no longer afford to deal with the problem of disability as a side line. We must deal with it as a business proposition rather than as a matter of sentiment or of social reform.

Few people are aware of the size of this problem and its social and economic implications. There are in this country 8,000,000 people who have physical disability of such nature and degree that at some time in their lives it will cause them difficulty either in getting or in holding a job. Of these, 6,000,000 are normally in employment, but 2,000,000 for one reason or another are unemployed. Although some of the latter get back to work, additional persons drop permanently out of the labor market. The pool is kept at a fairly constant level from a number of sources: (1) from among those currently becoming disabled in industrial, automobile, home and other accidents and from disease and congenital causes; (2) from among young disabled persons who reach employable age, and (3) for the duration of the war from among those disabled as a result of military operations. The annual increment from these sources is now at the rate of well over a million persons per year.

Thus, the minimum job of a rehabilitation service in this country is the rehabilitation of a million persons — to say nothing of another million from among the 6,000,000 who are normally employed but who drop out for one reason or another, such as progression of their disability, further injury or the acquisition of chronic disease and technologic changes in their jobs, and require rehabilitation in order to get back into employment. These 2,000,000 persons not only are not producing but are consuming approximately a billion dollars a year of the production effort of others.

The vast majority of people in this country earn their living through work; hence the ability of a person to work and to get a job represents the economic foundation of his life. Any condition which affects the economic foundation of 2,000,000 people affects the economic welfare of the nation. In normal times it is economic waste to support 2,000,000 nonproducers. In the present manpower situation it is sheer folly not to make them employable and put them to

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work as quickly as possible. It is a part of the present and the immediate future of rehabilitation to do just that.

After the war is over and we begin to pick up the pieces and to get our world put together again the job will have to be done over and under much more difficult conditions. The present program is wholly inadequate to the present task. It will be more so for the task ahead. I tell you frankly that unless the Congress sees fit to strengthen and extend the scope of the program, I see little or no future for it. If, however, the Congress does strengthen it and extend its scope, I see a great future for it. I see it as an important segment of social security for both the individual and the country.

The program which is now being considered by Congress would provide any service necessary to get an individual into a job and keep him there. It would be a comprehensive program, and it would be one strong enough to meet present and future needs. There are, however, two obstacles which must be removed if any rehabilitation program, present or future, is to fulfill its potentialities as a social instrument. The first obstacle is attitude.

In the past most employers have had the idea that a disabled worker is less efficient and more prone to accident than the physically normal and have taken on the handicapped only when physically normal workers were unavailable. They have failed to realize that a disabled worker who is properly matched with a job is a safer risk than a physically normal worker improperly matched with a job. They have used physical examinations for the exclusion of the physically imperfect rather than as one of the measures for the proper placement of workers in their plants. The present manpower shortage is compelling the employment of the handicapped, but the idea of using physical examinations, tests of aptitude, and personality inventories as a guide to putting the right man in the right job has not taken hold. This fact is resulting not only in the inefficient use of labor but in the injury and crippling of large numbers of urgently needed workers in our war production industries.

If employers would accept the principle of properly matching workers and jobs there could be no valid reason for the exclusion of handicapped workers — because a handicapped person is not handicapped in performing a job that is suitable to his physical condition, aptitudes and temperament. Obviously, there are large numbers of the handicapped who are physically unfit and should not be accepted for employment. However, most of them can be made fit through proper rehabilitative procedures. That is the job of the rehabilitation service.

We have a feeling that industrial surgeons should use their influence with management to obtain a more scientific method of placing workers in industrial plants and a more receptive attitude toward the employment of the handicapped.

The second obstacle is one about which I have addressed you on a previous occasion, that is the inequitable provisions of second injury clauses in workmen's compensation laws in most of the states. Most alleged workmen's compensation laws in this country are nothing more than employer liability laws, but some of them, while liberal to employers and insurance carriers, are positively vicious in their effects on those injured in industry and on the other taxpayers in the community. The medical benefits are farcially small, the disability benefits are insufficient and, worst of all, the second injury clauses serve as effective barriers to reemployment.

In several states a handicapped wroker has the choice of signing a waiver to his right to compensation in the event of a second injury or of not getting a job; in other states, where employers might be willing to hire handicapped workers, they cannot afford to do so because under their compensation laws they would be liable for the total results of a second injury — the cost of which in the event of permanent total disability would run into big money.

Do you see what these things mean to the injured worker and to the other taxpayer? They mean that, when the medical benefits are so small, the injured worker is obliged to expend a considerable part of his compensation benefits on medical care and when the remainder is gone his family or the taxpayers of his community must take care of him because the second injury clause in his state workmen's compensation law establishes an effective barrier in his going back to his job. Furthermore, in some states, although medical and compensation benefits are more nearly adequate, there is neither encouragement nor incentive for the injured worker to accept the opportunity of rehabilitating himself — nor is there any penalty for his failing to do so. The result is that many beneficiaries of workmen's compensation are pauperized.

My denunciation of the effects of these laws should not be interpreted as advocacy of their repeal. It is not. My plea is that they serve the purpose for which they were originally conceived — and I assure you that they were not conceived simply as a device for limiting the liability of the employer or insurance carrier. They were conceived as a social instrument first and foremost, for the conservation of the worker, and, second, as a means of distributing equitably the financial burden of industrial accident and disease.

The inequitable provisions of these laws in a number of states are seriously interfering with the recruitment of labor in war production plants. The legislatures of most of the states are now in session or will be during the next few months. May I urge that you interest yourselves and the employers with whom you may be associated in amending these laws in such a way that they will serve the purpose for which they were conceived. Let us make them real compensation laws, laws which will not only protect employers and compensate disabled workers but give incentive to the injured workers to accept the opportunity to rehabilitate themselves rather than to become pauperized. If this is done it will contribute now to the war effort, and it will greatly facilitate the rehabilitation of the thousands who are being injured in the war — a hundred thousand for each year of our participation in the war.

When I addressed you two years ago I made a proposal that you and the workmen's compensation and rehabilitation officials appoint committees to decide on a program for working together on the different problems in which we have a common interest, including the strengthening of workmen's compensation and rehabilitation legislation. I still think it would be a good idea.

I shall not repeat the proposal. I am, however, going to bring home to all of us the close community of interest we have in the matter, and how now in the future it will affect us personally and professionally. We are engaged in the most horrible war the world has ever seen. I imagine that many of us in this room have sons or daughters over there somewhere. Maybe they will come back safe and well — maybe they won't. If they should come back maimed or shattered in health we are going to want the best medical and rehabilitation services money can buy. Furthermore, we are going to resent any law or any attitude which will tend to discourage or prevent them from getting a job. We shall demand that they have a chance to participate in and enjoy the fruits of the democracy which they are fighting so valiantly to protect. They will have earned this chance, and if we who are the leaders in the fields of medicine, rehabilitation and workmen's compensation let them down by failing to do our best toward making it possible for them to work, who can blame them if they take things into their own hands and give themselves a new dispensation?

THE EFFECT OF THERAPEUTICALLY ADMINISTERED CARBON DIOXIDE INHALATION ON THE RESPIRATION IN PULMONARY TUBERCULOSIS *

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We have been using carbon dioxide by inhalation in the treatment of pulmonary tuberculosis since 1930 and reported some of our observations in previous publications.[1] The inhalations were given on the basis of the following indications: 1. Strenuous, exhaustive cough without effective expectoration if there is evidence of inflammatory exudation in the lungs; when the cough is insufficient to evacuate sticky, thick sputum and thereby causes gagging, vomiting or incontinence, or when the sleep or rest of the patient is disturbed by coughing. 2. Dyspnea originating from lobular atelectasis due to bronchial obstruction caused by tuberculous involvement of the bronchial wall or by viscid mucopurulent plugs. 3. Dyspnea originating from massive atelectasis. 4. Presence of large cavities with insufficient drainage through the connecting bronchus. 5. Bronchiectasis secondary to pulmonary fibrosis associated with the healing of a tuberculous process when postural drainage is impractical or is not effective for evacuating the stagnating secretions from the dilated bronchi. 6. For aiding expectoration in cases of retention of inflammatory products following phrenic nerve block or during the course of artificial pneumothorax. 7. Cough which is not strenuous but is not sufficient to remove accumulated material from the lungs.

Review of the Literature

Miescher[2] in 1885 called attention to the importance carbon dioxide plays in respiration and formulated the aphorism: "Over the oxygen supply of the body carbon dioxide spreads its protecting wings." Haldane and his associates[3] demonstrated experimentally that an increase of the carbon dioxide in the pulmonary aveoli was consistently accompanied by an increase of the respiration. It was found that the inhalation of as low as 1 per cent carbon dioxide stimulates the respiratory center and causes deeper breathing. Hill and Flack[4] reported that the inhalation of increasing concentrations of carbon dioxide caused a proportional stimulation of the respiratory center until 10 per cent was reached; higher concentrations led to a progressive depression of respiration. Brown[5] found that the maximum stimulation of the pulmonary ventilation was reached at 10.4 per cent carbon dioxide. The greatest protagonist of the clinical application of carbon dioxide therapy was Henderson,[6] who with Haggard[7] and other associates made numerous contributions to the medical literature on the practical value of this method of treatment. They recommended the inhalation of 5-7-10 per cent carbon dioxide for the treatment of carbon monoxide poisoning and of

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asphyxia from various causes. Its use was also advocated for the resuscitation of newborn infants, for the prevention and treatment of surgical shock during major operations and for the prevention and treatment of postoperative massive atelectasis and pneumonia. Henderson found that the inhalation of carbon dioxide is an effective means of controlling persistent hiccough and the paroxysmal stage of whooping cough and for alleviating the consequences of the withdrawal of morphine in addicts. Also, he recommended its use in patients with effort angina. Hunter and Mudd[8] and Robinson and Selesnick[9] produced prompt recovery in patients in alcoholic coma by the inhalation of a mixture of carbon dioxide and oxygen. McConnell and McCormack[10] used carbon dioxide inhalation in their obstetrical practice and arrived at the conclusion that carbon dioxide and oxygen mixtures safely and promptly combated uterine inertia in the first stage of labor and controlled postpartum hemorrhage; furthermore that these inhalations were effective for the relief of asphyxia neonatorum and atelectasis neonatorum and of abdominal distention following cesarean section. Carbon dioxide by inhalation was administered successfully by Hurst[11] in the treatment of bronchial asthma and by Alison[12] in the treatment of bronchitis. Encouraging results were obtained by Christie[13] in cases of unresolved pneumonia.

Pharmacology

It is generally accepted that short inhalations of 5 to 10 per cent carbon dioxide with ample oxygen induces no ill effect. The most conspicuous manifestation of their influence is an increase in the respiratory minute volume. In a self experiment on one of us (A. L. B.) with 10 per cent carbon dioxide in 90 per cent oxygen, 6 liters being taken per minute through a face mask, the respiratory rate rose from 14 per minute before the inhalation to 19 at the end of the experiment: Fifteen minutes. Henderson, using 5 per cent carbon dioxide, found that the volume of breathing increased more than threefold. Also, marked augmentation in the respiratory minute volume was recorded by Heller and his associates,[14] Barcroft and Margaria,[15] Hitzenberger[16] and Grueneberg and Viethen.[17] The effect usually becomes noticeable during the first minute of inhalation. Brief interruptions render more sensitive the respiratory center. Carbon dioxide causes one to breathe not only more per minute but also with fuller lungs. It quickens the rate both of the air inhaled and of the air exhaled. There are considerable variations in the same person on different days. Some persons show only slight increase in minute volume to 5 per cent, even to 7 per cent carbon dioxide.

Hartl[18] found that oxygen intake is increased at the beginning of the inhalations, it may remain the same or may decrease during the treatment. Saklad[19] observed that when normal oxygenation was accomplished in anoxemic patients, the respiratory rate diminished. The experimental studies of Prinzmetal[20] revealed that the increased respiratory motions of the chest during carbon dioxide inhalations are associated with a greater negativity of the intrapleural pressure on inspiration and a reduced negativity on expiration: both these changes contribute effectively to a competent diastole and systole of the lung. Henderson[6] postulated an increase in the tonus of the respiratory muscles of the chest, particularly the diaphragm, and of the myoelastic structures of the lung during carbon dioxide inhalations. The latter assumption was proved to be correct by the clinical observations of Brunn and Brill.[21] They studied the effect of carbon dioxide during bronchoscopy and noted that it induced violent movements of the bronchial tree and alterations in the shape of its branches: consequently bronchial secretions spilled from minor into major brochi.

The inhalation of 1 to 8 per cent carbon dioxide improves the functional capacity and the ventricular output of the heart;[17] also it increases both the systolic and the diastolic blood pressure[22] and is followed by an increase in the pulse rate and in the pulse pressure, particularly after the concentration of carbon dioxide has reached 3 per cent.[23] Tomaszewski and his coworkers[24] administered 8 per cent carbon dioxide to healthy persons, and they found, besides the respiratory changes, a slight increase in the metabolism and an initial quickening and then slowing of the pulse. No increase in the metabolic rate was noted by other investigators.[25] Fuchs[26] observed that a slight increase in the blood carbon dioxide accelerated clotting, while too much carbon dioxide in the plasma caused prolonged coagulation. Tannenberg[27] considered this accelerated clotting an indirect transient effect of carbon dioxide on the peripheral and central nervous system that leads to an enlarged production of epinephrine.

Clinical Study

The data presented in this paper are based on observations made on 40 patients with moderately advanced and far advanced pulmonary tuberculosis. All of them were given carbon dioxide oxygen inhalations as a therapeutic measure for the treatment of otherwise uncontrollable cough and dyspnea. The apparatus used in our work consists of a tank, mounted on a small platform with casters, containing a mixture of 10 per cent carbon dioxide and 90 per cent oxygen. A reducing valve and oxymeter serve to regulate the flow of gas per minute. A rubber bag which serves as a small reservoir is connected to the tank. The patient was seated on a chair, and the technic and purpose of the procedure was explained to remove fear or apprehension. After the determination of the respiratory rate and pulse rate in this position, from two to five counts were taken during a ten to fifteen minute period of inhalation of the gas mixture. Ten of the 40 patients took the treatments with the aid of a face mask, by the closed method; the others inhaled the gas through a glass tube. In the latter cases the patient was instructed to hold the end of the glass tube in his mouth, to inhale the gas coming from the tank and to exhale through the nose. Although the admixture of air with and dilution of the inhaled gas was unavodiable, judging from the therapeutic response this method of administration proved to be satisfactory. Altogether 275 observations were analyzed: 17 treatments by the closed method and 258 treatments by the open method (through a glass tube).

It was readily noticeable that under the effect of inhalations all patients were breathing deeper and with a fuller chest. During the treatment they felt comfortable, although they were conscious of taking deeper breaths. Their comments as to the immediate effect were impressive: "The breathing became easier," "the chest feels lighter and clearer," "the inhalations act as if they were loosening up the lungs." The therapeutic results following these treatments were: (1) easy and effortless expectoration; (2) diminution of cough and (3) relief from dyspnea.

By the closed method the amount of gas delivered to the patient varied from 4 to 6 liters per minute. The anticipated response to such high concentrations of carbon dioxide was an increase in the respiratory rate. Still, we found this in 3 instances only. The respiratory rate remained unchanged in 3 and decreased in 9 observations. This is at variance with findings in normal persons and can be explained (1) by the reinflation of previously atelectatic areas of the lung with a consequent immediate relief from dyspnea and (2) by the increase in the amplitude rather than in the rate of respiration. In 2 instances the rate dropped in four minutes from 26 to 14

and from 24 to 10, respectively. In 2 patients an initial decrease in the rate was followed by a moderate increase toward the end of the treatment.

With the open method the pattern of respiratory response was similar to that found with the closed method and is presented in tables 1, 2 and 3.

TABLE 1.

Respiratory rate at end of inhalations	$O_2 + CO_2$ Liters Per Minute: 4 No.	%	4½ No.	%	5 No.	%	5½ No.	%	6 No.	%	6½ No.	%	7 No.	%	Total No.	%	Total No.	%
Unchanged	3	37.4	1	11.1	55	30.4	..	.....	3	33.3	4	44.4	10	25.6	76	29.5	76	29.5
With direct decrease	1	12.6	4	44.4	53	29.3	2	66.6	2	22.2	2	22.2	19	48.7	83	32.2	....	.....
Decreased —																	93	36.0
With initial increase	.....	.....	..	.....	9	4.9	..	....	..	.....	..	.....	1	2.6	10	3.8	....	.....
With direct increase	2	25.0	2	22.2	51	28.2	1	33.3	3	33.3	2	22.2	3	7.7	64	24.8	....	.....
Increased —																	89	34.5
With initial decrease	2	25.0	2	22.2	13	7.2	..	.....	1	11.1	1	11.1	6	15.4	25	9.7	....	.....
Total	8	100	9	100	181	100	3	100	9	100	9	100	39	100	258	100	258	100

TABLE 2.

Respiratory rate at end of inhalations	Frequency of Intermediate Changes During Inhalations: Number	Per Cent	Total Number	Total Per Cent
Unchanged —				
No intermediate change	34	44.7	....	.....
With intermediate decrease	27	35.5	76	29.5
With intermediate increase	15	19.7	....	.....
Decreased —				
No intermediate change	52	55.9	....	.....
With intermediate increase	31	33.3	93	36.0
With initial increase	10	10.8	....	.....
Increased —				
No intermediate change	58	65.2	....	.....
With intermediate decrease	6	6.7	89	34.5
With initial decrease	25	28.0	....	.....

TABLE 3.

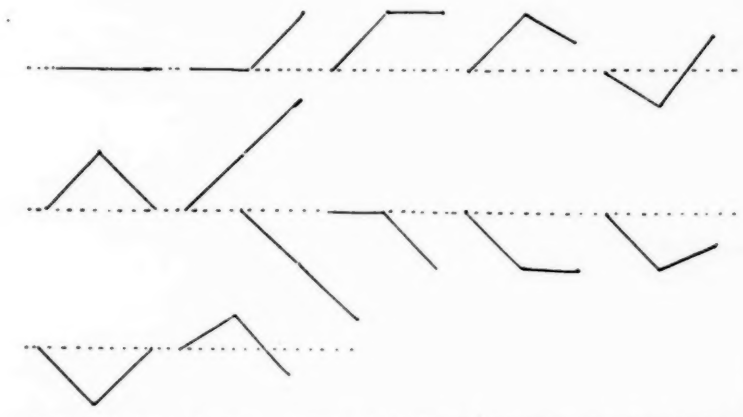
Change in respiratory rate	Extent of Deviations from Initial Respiratory Rate: Less Than 10% No.	%	10-19 No.	%	20-29 No.	%	Per Cent 30-39 No.	%	40-49 No.	%	50-60 No.	%	Inhalations No.	%
Decrease —														
Final rate	38	40.8	41	44.1	12	12.9	1	1.1	1	1.1	..	....	93	100
Intermediate and initial deviations	15	37.5	16	40.0	4	10.0	3	7.5	2	5.0	..	....	40	100
Increase —														
Final rate	24	27.0	26	29.2	26	29.2	4	5.4	2	2.7	1	1.4	74	100
Intermediate and initial deviations	20	27.0	22	29.7	25	33.8	9	10.1	3	3.3	1	1.1	89	100

Of the 258 observations with the open method, 4 liters of the gas mixture per minute were given in 8 instances, 4, 5 in 9; 5 in 181; 5.5 in 3; 6 in 9; 6.5 in 9 and 7 in 39.

At the end of the inhalations the number of respirations was increased in 89 instances. The increase varied from less than 10 per cent to 60 per cent of the initial rate; the great majority showed a rise less than 30 per cent. In 93 instances the number of respirations was decreased; the reduction varied from less than 10 per cent to 42.6 per cent; it was less than 20 per cent in 79 instances.

The initial respiratory rate varied between 16 and 40; the great majority were between 20 and 29. There was a group of observations in which during the inhalation of carbon dioxide the respiratory rate first decreased and then increased; in another group an opposite response was recorded.

In cases in which the rate at the end of the observation period was the same as the original rate, there was an intermediate decrease in 27, or 35.5 per cent, and an intermediate rise in 15, or 19.7 per cent. In the group with a final reduction in the respiratory rate an intermediate increase was noted in 31, or 33.3 per cent. In cases in which the final respiratory rate was elevated, an intermediate decrease was seen in 6, or 6.7 per cent. The intermediate increase varied from less than 10 per cent to 60 per cent of the



Pattern of changes in the respiratory rate during the inhalation of carbon dioxide as observed at ten and fifteen minutes intervals, compared with the preinhalation rates.

initial rate, the great majority showing a rise between 10 and 29 per cent. The intermediate decrease varied from less than 10 per cent to 42.6 per cent of the initial rate, the great majority falling between 6 and 19 per cent.

It is interesting to note that the final respiratory rate was the same as the initial rate in 76 instances, or 29.5 per cent, and it was less than the initial rate in 93, or 36 per cent. Altogether, the final rate remained unchanged or was reduced in 169 instances, or 65.5 per cent, as against 89 instances, or 34.5 per cent, in which the respiratory rate was increased at the end of the inhalation of the gas mixture.

Conclusion

1. In connection with the clinical use of carbon dioxide inhalations in our practice we have made observations concerning the effect of carbon dioxide upon the respiration of patients with moderately advanced and far advanced pulmonary tuberculosis. A group of 40 patients was studied and 275 observations were recorded.

2. A mixture of 10 per cent carbon dioxide and 90 per cent oxygen was administered either by the closed method, through a face mask, or by the open method, through a glass tube, the end of which was held in the patient's mouth.

3. It was readily noticeable that carbon dioxide administered by either method acted as an effective stimulant of the respiratory center and induced an increased amplitude of the respirations.

4. A decrease in the respiratory rate was the most frequent response to inhalations by the closed method.

5. When the open method was used and the mixture was diluted with the inhaled air, the final respiratory rate was the same as the initial rate or decreased in 65.5 per cent and increased in 34.5 per cent.

6. The decrease in the respiratory rate is attributable: (*a*) to the augmented respiratory expansion of the chest; (*b*) to the opening up and reventilation of atelectatic areas of the lung and (*c*) to adequate oxygenation of the blood.

7. In tuberculosis of the lungs the administration of a respiratory stimulant such as carbon dioxide may appear paradoxic at first glance in view of the induced increase in respiratory expansions. But our experience shows that the short periods of augmented inspiratory motions of the lung are far outweighed by the therapeutic benefits derived from them, namely (*a*) diminution of cough; (*b*) easy, effortless expectoration and (*c*) relief from dyspnea, with consequent sustained rest for the diseased lung.

8. The analysis of our protocols is presented for the purpose of illustrating that the inhalation of a mixture of 10 per cent carbon dioxide and 90 per cent oxygen by the methods described is a safe procedure that does not interfere with the comfort of the patient; consequently it can be applied as a useful clinical method for the management of cough and for the treatment of dyspnea due to atelectasis in selected cases of pulmonary tuberculosis.

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THE CONTROL OF THE PERIPHERAL CIRCULATION

A Review of the Physiologic Literature

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The regulation of the peripheral circulation offers an extremely complicated physiologic problem. The peripheral blood flow is the sole supply and the main drainage for the local metabolism. It controls the temperature of the tissues and is essential in the maintenance of the temperature of the whole body. Moreover, it is essential for the regulation of blood pressure and of the whole cardiovascular system. The physiologic range of requirements on the peripheral circulation is tremendous. The metabolism of a working muscle is many times its resting level. Circulatory adjustments during a change from a reclining to an upright position are considerable. Body temperature remains constant under greatly varying conditions of heat production and loss.

The demands on the peripheral circulation may be excessive under pathologic conditions, and it may fail, as in shock, in fever and in a shaking chill. Abnormal vascular reactions appear in many diseases, e. g., arthritis and peripheral vascular disease. The approach to therapeutic measures such as sympathectomy ought to be based on the physiology of the peripheral circulation.

A complex system of controls adjusts the peripheral blood flow to meet physiologic and pathologic requirements. The following review will be limited to direct adjustments of blood vessels. It will not consider factors which influence the heart and thereby the circulation as a whole. The structure of vascular regulations consists of three parts, namely nervous, humoral and local chemical or physical factors. Of course, these three divisions are actually linked together in an inseparable unit. The nervous sys-

tem acts by the local release of chemical transmitter substances. The same substance is frequently a local vaso-active metabolic and a humoral factor affecting the distant blood vessels; or a transmitter of nervous impulses is simultaneously a humoral agent.

The innervation of the blood vessels is vasoconstrictor and dilator.[1] It involves the whole arterial tree, the capillaries, the arteriovenous anastomoses and the veins.[2] However, separate reactions of these divisions are apparently possible, e. g. in isolated arteriolar constriction or capillary dilatation. The presence of muscle fibers in capillaries and arteriovenous anastomoses is doubtful, and nervous impulses may have another mode of action than on larger vessels.[3]

The highest vasomotor centers appear to be in the hypothalamus and the lower ones on the floor of the fourth ventricle and in the cervical section of the cord. Vasoconstrictor and dilator centers are seemingly separate units.[1] Constrictor impulses are carried by sympathetic fibers leaving the cord with the thoracolumbar outflow. After the ganglionic transfer they reach the blood vessels via the splanchnic nerves, via sympathetic fibers in mixed peripheral nerves and (on the neck and head) via perivascular networks. The vasodilator nerves present an anatomic problem which is still under discussion. Dilator fibers are present in parasympathetic nerves, such as the chorda tympani. Parasympathetic dilator fibers travel with sympathetic nerves.[4] Sympathetic fibers themselves are capable of transmitting dilator impulses under certain conditions. Finally, dilator impulses seem to find an antidromic passage through posterior roots and sensory nerves.[5]

The blood vessels of an inactive organ, e. g. a muscle, are not completely relaxed; they show a certain vasomotor "tone." The normal tone is at least partly maintained by continuous nervous impulses. The vasomotor centers regulate the vascular tone. When the requirements of an organ grow with activity the constrictor tone of the blood vessels is released and dilator impulses are transmitted to increase the local blood flow. Simultaneously vasoconstriction appears in resting organs. The available circulating blood volume is thereby economically utilized and the systemic blood pressure is maintained.

The vasomotor centers must therefore receive and react to numerous afferent stimuli from all parts of the body and release innumerable constrictor and dilator impulses of varying intensity to all parts of the body. Higher brain centers affect the hypothalamic vasomotor apparatus, as proved by electrical stimulation or by emotional flushing and pallor.[6] The temperature-regulating centers of the hypothalamus function largely with the aid of the vasomotor areas. On receiving cold stimuli by nerve routes and decreased blood temperatures the hypothalamic centers effect a constriction of skin vessels and a dilatation of muscle vessels.[7] The peripheral stimulus of heat has a more or less reverse influence on temperature and vasomotor centers and the circulation.

The aorta and carotid arteries are the origin of afferent nerves which register pressure changes in these large blood vessels.[8] An increase in pressure for whatever reason or electric stimulation of the depressor nerves produce peripheral vasodilatation via vasomotor centers. A fall of pressure in the central vessels leads to peripheral vasoconstriction. Similar reflexes originate in the large veins[9] and in mesenteric vessels.[10] As a matter of fact, afferent impulses may arise from almost any part of the body and cause reflex vasomotor changes.[11] Strong stimuli, especially if painful, result as a rule in peripheral vasoconstriction, while brief or mild excitations fre-

quently cause dilatation. These vasoconstrictor effects appear mainly in skin and splanchnic vessels in the presence of an unchanged or even increased blood flow in the muscles. Chemical receptors[12] such as the carotid body send nervous impulses mainly to the respiratory centers but exert some influence also on vasomotor centers. The carotid body reacts especially to lack of oxygen or poisons, such as cyanide. The vasomotor centers are very sensitive to certain humoral influences, chiefly the carbon dioxide content of the arterial blood.[13] Hypercapnia stimulates the constrictor mechanism; hypocapnia diminishes the vascular tone. Acidosis and anoxemia have an effect similar to (but less intensive than) that of hypercapnia.

The impulses of constrictor nerves are transmitted to the blood vessels by the local release of epinephrine-like substances or epinephrine proper.[14] Dilator effects are produced by the liberation of acetylcholine, e. g. from the chorda tympani and probably from antidromic dilator fibers. However, physiologically small amounts of epinephrine given off from true sympathetic nerve endings may also have a dilator action.[14]

Whether sympathetic stimulation results in vasoconstriction or dilatation does not depend only on its intensity and duration and thereby on the quantity of released epinephrine; the effector organ and its momentary metabolic condition play a major role in the type of vasomotor response.

Blood vessels of the skin react to sympathetic stimulation or the injection of epinephrine as a rule with contraction. Even in the skin, however, different areas show different reactions, at least to reflex vasoconstriction, for example the forehead and forearm as compared with the fingers. The response of large skin arteries is not the same as that of arterioles and capillaries.[15] Splanchnic blood vessels constrict under strong stimuli and dilate under small doses of epinephrine. They always dilate if the constrictor action of the sympathicus (and of epinephrine) is blocked by an injection of ergotamine. About ten times as much epinephrine is needed to produce constriction of kidney vessels as elsewhere.[16] The sympathicus and epinephrine have only a dilating effect on the coronary vessels. Similarly, many investigators believed the sympathicus to be a vasodilator for the muscle. Recent experiments proved, however, at least for the dog under anesthesia, that sympathetic stimulation and epinephrine constrict the blood vessels of the resting and dilate those of the contracting muscle.[17] Constrictor reflexes originating from the carotid sinus or from an accumulation of carbon dioxide in the blood are also ineffective on a contracting muscle or during reactive hyperemia.

Vasomotor regulations are further complicated by the fact that epinephrine—the peripheral local transmitter of the sympathetic stimulus—is released by the chromaffine system into the blood stream. As a humoral agent it produces in the whole circulatory system effects which vary according to hormonal concentration and the regional vascular responsiveness. Moreover, on reaching the sympathetic chain from the blood stream, epinephrine in small physiologic doses facilitates the transfer of impulses in the synapse.[18] Thereby it increases greatly—as a humoral factor and indirectly—the vasoconstrictor effect of preganglionic stimulation (and of vasoconstrictor reflexes?). Epinephrine impedes the synaptic transfer in larger physiologic concentrations. The transfer of the sympathetic excitation within the synapse occurs through the local release of acetylcholine. Epinephrine enhances the action of acetylcholine in the sympathetic synapse in small doses and interferes with it, even irreversibly, in larger doses.[18] This last effect might help to explain the mechanism of epinephrine shock.

Another vasoconstrictor hormone is produced by the posterior pituitary

lobe. It is supposed to act on capillaries mainly, but its physiologic role has not yet been sufficiently studied.[19] Similarly, the significance and action of humoral vasoconstrictors produced by the anoxic and by the normal kidney need much clarification. Apparently the kidney contains and releases during hypoxemia a ferment-like factor, renin, which in contact with serum globulin forms a pressor-vasoconstrictor substance. There are more known humoral vaso-active agents, but they act primarily in the area of their liberation and will therefore be mentioned later.

Humoral vasoconstrictor factors probably play a role in the maintenance of vascular tone. After postganglionic or preganglionic denervation blood vessels regain their tone in a few days and frequently exhibit in eight to ten days an abnormal increase, which gradually diminishes to a more or less normal level.[20] Like all other effector organs, blood vessels develop after denervation a hypersensitivity to numerous stimuli and overreact at least temporarily to humoral constrictors such as epinephrine. The hyperexcitability is more marked in a postganglionic than in a preganglionic lesion.

The nervous and humoral regulations of the blood vessels are responsible for the coordination of the whole circulation and mainly activate constrictor reactions. When the peripheral tissues need a greater blood supply, powerful local vasodilator mechanisms spring into action. A muscle when contracting suddenly requires a five to ten times greater blood flow than it required when at rest.[21] A number of the metabolites produced during the contraction have vasodilator effects. Carbon dioxide, which stimulates the vasoconstrictor centers, is a powerful peripheral dilator, and lactic as well as other acids have a similar local action.[22] The dilatation probably takes place on the minute vessels and is followed by vasovascular (or axon) reflexes.[23] These result in dilatation of the inflow arteries and a true active hyperemia. Adenosine triphosphoric and monophosphoric acids are other muscle metabolites of dilator character.[24] Increased amounts of histamine have been found in the venous blood of a contracting muscle.[25] Histamine is a capillary dilator, and the dilatation spreads by means of axon reflexes to inflow arterioles. Histamine-like substances are probably responsible for almost any type of skin hyperemia. Potassium is released by the muscle during contraction and during anoxia. It has a local vasodilator action. If it enters the blood stream in sufficient concentration, however, it stimulates the chromaffin system to release epinephrine, with a resulting humoral and systemic vasoconstriction and pressure action.[26]

Physical factors such as heat and cold have a local dilating or contracting influence on minute and larger blood vessels.[27] Partly it may be caused by the local release of vaso-active metabolites, discussed in the previous paragraph. Rapidly appearing changes in the vascular caliber are, however, actually vasomotor reflexes, while direct vasoconstriction by cold is slow in developing. Local constriction on exposure to intensive heat and intermittent dilatation under severe cold[15] are obviously of reflex nature. The reactive hyperemia following cold application is supposedly caused by local vasodilating metabolites.[15] Ultraviolet radiation and other forms of radiation energy produce erythema supposedly by the formation of fixed histamine-like substances in the skin.[28]

Although no details of the actions and the interplay of the vaso-active metabolites are known, it is certain that active tissues take care of their greater blood requirements mainly by direct, local vasodilatation. The indirect humoral and the local tissue effects of potassium, and similarly the central and peripheral effects of carbon dioxide, are seemingly antagonistic. Physiologically this represents a most effective coordination of actions. The

same substance causes vasodilatation where hyperemia is required and elsewhere a vasoconstriction important for the maintenance of the blood pressure and for an economic shift of the circulating blood volume. Moreover, the local metabolic vasodilatation remains unaffected by most central or humoral vasoconstrictor stimuli. Hypercapnia, depression of the carotid sinus, stimulation of the sympathetic system and injection of epinephrine are incapable of producing vasoconstriction in a contracting muscle or in one that is in reactive hyperemia.[29] Suffocation or inhalation of very high amounts of carbon dioxide will overwhelm local vasodilatation. Human skin in reactive hyperemia responds to a topical application of epinephrine with constriction. As a rule, however, metabolic hyperemia is not interfered with by the vasoconstrictor reactions which simultaneously adjust the circulation as a whole.

The kidney seems to have an exceptional position within the system of blood flow regulations. Over a wide range of systemic blood pressure changes the kidney maintains its "private" circulation at a constant rate by means of local vascular adaptations. It does not share the endless readjustments which take place elsewhere in the interest of the general circulatory management.[16] It reacts only to very large doses of epinephrine. The circulation of the brain, heart, lungs and liver offer special problems which cannot be discussed here.

While tissue metabolism has a predominant influence on the local blood supply, the circulation has possibly some effect also on the local metabolism. Vasomotor constriction can reduce the blood flow in an organ to a marked degree without causing reactive hyperemia.[30] The same diminution of blood flow by mechanical obstruction will result in dilatation of the minute vessels and after release of the compression in reactive hyperemia. Vasoconstriction caused in the limb of an anesthetized dog by carbon dioxide inhalation, depression of the carotid sinus, sympathetic stimulation or injection of epinephrine leaves the arteriovenous oxygen difference unchanged or decreases it. Mechanical reduction of blood flow to the same degree increases the arteriovenous oxygen difference. In the presence of an equally diminished arterial inflow and a complete oxygen saturation of the arterial blood in both instances, the unchanged venous oxygen content in vasomotor constriction points to lower oxygen consumption of the tissues, and the lower venous oxygen in arterial compression points to an unchanged oxygen uptake of the tissues. It appears, therefore, possible that vasomotor reactions control to some degree the metabolism of involved tissues. The sympathicus is supposedly the carrier of these impulses. An attempt to explain the observations during vasoconstriction by shunting of blood through arteriovenous anastomoses does not stand up under criticism.[31]

Even this brief and incomplete physiologic review should be impressive enough to indicate the great difficulties in approaching the problems of the pathology or therapy of the peripheral circulation. Obviously the control of the peripheral blood flow can break down in numerous ways to produce the syndrome of shock: excess release of epinephrine, deficient production of epinephrine, excess and systemic overflow of vasodilators such as histamine, damage to vasomotor centers, excess dilatation in large vascular areas such as the skin, or any combination of these factors, and in addition traumatic destruction of large districts of capillaries, hemorrhage and acute heart failure. With such varying mechanism, the main clinical features and effective therapy must vary, too. Total or extended sympathectomies will necessarily interfere with the important systemic adjustments of the peripheral circulation in addition to producing local maladjustments of denervated

blood vessels, possibly caused by their hyperexcitability. Nerve section for pain in peripheral vascular disease or sympathectomy may be complicated by a loss of nervous control of tissue metabolism. Much work will have to be done to clarify these and many other problems of the peripheral blood flow.

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THE PHYSICAL THERAPY OF PERIPHERAL VASCULAR DISEASE *

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The study of peripheral vascular diseases has advanced by leaps and bounds in the past decade, and the medical literature is replete with articles on various phases of this subject. As is true of every disease for which there is no specific therapy, the treatment measures in this field are many; but, because of the inherent nature of most peripheral vascular diseases, physical therapy is the most favored of these measures. Numerous appliances and complicated machines have been developed by enterprising investigators. Many of these are merely improvements of or combinations of older devices. The pavaex boot, developed by Herrmann and Reid[1] in 1933, is a modification of the glass boot devised by Junod in 1834, and used at that time for the application of suction to a diseased extremity. The practice of intermittent venous occlusion, so widely used today by means of the apparatus of Collens and Wilensky and other types of venous constrictors, is based upon the principle expounded in 1903 and known as Bier's hyperemia.

Innovations are so constantly being made that there is available today a bewildering array of physical therapy devices. The therapeutic claims of the manufacturers of each instrument are supported by bona fide case reports prepared by creditable investigators. What then is the average physician to do who is called upon to treat peripheral vascular diseases? He must make a choice from many diverse machines, all of which are relatively costly. Or, take the case of the clinic which is fortunate enough to have several types of such apparatus: How shall the treatment program be apportioned between them? Which is better adapted for the treatment of this or of that condition?

Our purpose is broadly to summarize our opinion of some of these apparatus, as derived from 3 years' experience in the treatment of peripheral vascular disorders; and to outline a rational program for the physical therapy of those disorders.

It is axiomatic that by mechanical means alone it is impossible successfully to treat diseases of the arteries. Physical therapy is but one of the conservative approaches to this problem. Medical management is of equal importance. Surgery, too, in its minor phases, has its indications in this field. As important as the realization of the limitation of physical measures is appreciation of the fact that, fundamentally, this group of diseases can be classified into two broad categories—functional and organic. The therapeutic management varies greatly in these two groups. In the functional types the goal is to overcome vasospasm or vasodilatation, as the case may

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be; while in the organic type the treatment aim is to repair the organic insult to the arterial pathways and to foster the development of collateral channels. It must be emphasized that many of the cases which we are called upon to treat are combinations of functional and organic conditions; and, without accurate diagnosis, one cannot properly prescribe the appropriate treatment, nor evaluate the results with any certainty.

Suction and Pressure

Among the devices employed in the clinic at Veterans' Administration Facility, Pittsburgh, is the suction and pressure apparatus, which operates on the principle of Herrmann and Reid's pavaex boot. This machine consists of a motor, a pump, and four boots made of cellulose acetate, into which the extremities are inserted and sealed by means of a rubber cuff to make the unit airtight. The pump, driven by a motor, alternately compresses and decompresses the part in the boot. Landis and Gibbons[2] who made some of the pioneer studies in this field and who, incidentally, built their own apparatus for alternating positive and negative pressures, utilized Poiseuille's law, namely, that the amount of fluid which will flow through a rigid tube depends on the fall in pressure along the tube. They believed that it should be possible to reduce capillary and venous pressures in an extremity by applying suction. The full effect of such suction can be obtained only in the presence of a reservoir to accommodate the blood which has flowed through the narrowed and diseased arteries. However, capillaries and veins will serve only a limited capacity. By applying suction and pressure alternately, the capillary and venous reservoirs are filled and emptied in turn. The treatment is in this way designed to improve the circulation, carry more blood to the impoverished tissues, and encourage the development of a collateral blood supply.

Since Herrmann and Reid[3] gave the initial impetus to this form of therapy a controversy has raged as to the efficacy and applicabiltiy of the suction and pressure apparatus. Those investigators claimed excellent results in a wide variety of conditions. The enthusiasm of that day is reflected in the prediction of Shipley and Yeager[4] that suction and pressure "has great possibilities as a therapeutic measure, not only in thrombo-angiitis obliterans, arteriosclerosis, and various occlusive diseases, but also in ununited fractures, arthritis (atrophic and hypertrophic), frostbite gangrene, and the various circulatory disturbances that result from trauma." Horton, Krusen, and Sheard[5] concluded in 1939 that the suction and pressure method is not capable of promoting increased blood flow in normal extremities, and is of no value in the treatment of peripheral vascular disease. They based their opinion on the failure of the method consistently to raise the skin temperature of treated extremeties. But de Takats, Beck, and Roth,[6] questioning that criterion, concluded in turn that the clinical improvement which they have observed demonstrated the merit of the suction-pressure apparatus. Many other investigators have published their experiences with this device, and opinions are still divided. Allen and Brown[7] do not recommend its use. Among those who find it useful, although with reservations in some instances, are Collens and Wilensky,[8] Silverglade,[9] Beardwood, Jr.,[10] and Theis.[11] The latter reports encouraging results in frostbite.

It is apparent, then, that the initial enthusiasm which greeted the introduction of this mode of therapy has slowly been replaced by a reappraisal of its limitations and advantages. It is generally agreed that its value in frostbite, acute arterial embolism, and thrombosis is considerable. However, in other obliterative diseases, such as arteriosclerosis and throm-

boangiitis obliterans, its usefulness has been largely discounted. We have had excellent results in the treatment of frostbite gangrene. In several cases of arterial thrombosis and fairly recent arterial embolism, the symptomatic and objective improvements has been most gratifying. Notwithstanding many published reports to the contrary, we have concluded that in certain cases of arteriosclerosis obliterans and thromboangiitis obliterans, the relief of subjective symptoms, healing of ulcers, and reappearance of healthy color and warmth to the treated extremity attest to the merit of suction and pressure. (See table 1.) We have been very careful in selecting the patients

TABLE 1. — *Results of Treatment of Peripheral Vascular Diseases With Suction and Pressure as the Chief Mechanical Method.*

Diagnosis	Number of Cases Treated	Average Hours of Treatment	Number Improved	Percentage Improved
Arteriosclerosis obliterans	19	64.5	15	79
Arteriosclerosis obliterans with diabetes	14	59.3	13	92
Thromboangiitis obliterans	27	31.1	18	66
Frostbite	4	106	4	100
Arterial embolism (recent)	1	21	0	0
Arterial embolism (residuals of)	2	87.5	2	100
Arterial thrombosis (undetermined etiology)	2	57.5	1	50
Enderarteritis obliterans (syphilitic)	1	60	1	100
Aneurysm, popliteal (post-operative)	1	45	1	100
Polycythemia vera, with arterial thrombosis	2	34	2	100

for this treatment, excluding those in which any of the well-known contraindications exist. In our experience, no patient's pathological condition was aggravated by suction and pressure, although all of our patients did not obtain the improvement which was desired.

The degree of suction and pressure and the duration of treatment have been varied by us to meet the needs and indications of the individual. The average fluctuation of pressures employed have ranged from 80 mm. of suction (negative pressure) to 20 mm. of pressure (positive pressure). There are two general types of curves which can be plotted to illustrate the rise and fall of pressure in most devices of this kind. The one consists of a rapid fall from positive to negative, and vice versa. The other describes a slow fall and rise of pressure similar to a slow sine wave. Some attempt has been made to attribute certain advantages to the one or the other, but these claims are apparently without much basis. Our apparatus is of the rapid rise and fall type.

In administering this therapy we give a minimum of 1 hour and as much as 8 hours daily in selected cases. It is illogical to set a general maximum or minimum number of hours of treatment for any group of peripheral vascular diseases. The treatment is carried out in a room whose temperature is maintained at about 85 F. The patient keeps each of his hands on a hot water bottle for the duration, to produce reflex vasodilatation. With the environmental temperature at 85 F., vasospasm in the involved extremities is eliminated. Environmental temperature is of greater importance in influencing blood flow to the extremities than are all the other various factors known to exert such influence. A word of caution is necessary at this point. The ability of a diseased part to cope with increased environmental temperatures diminishes as the degree of vascular impairment increases. An ischemic extremity is unable to cope with the increased metabolic demand produced by excessive heat (about 100 F.), thus furthering the possibility of gangrene.

Intermittent Venous Occlusion

In 1936 Collens and Wilensky[12] reported good results in the treatment of 29 cases of peripheral obliterative diseases by a new method, intermittent venous occlusion. Since then several modifications of this apparatus have been developed, and considerable controversy as to its efficacy has arisen. The device was accepted by the Council on Physical Therapy of the American Medical Association on the grounds that, from the physiologic viewpoint, the production of intermittent venous hyperemia to increase peripheral circulation seems sound; and that tests indicate that both surface temperatures and the oscillometric curves increase after the application of this method. During venous occlusion there is a filling and stretching of the venocapillary bed, while during release a reactive hyperemia takes place, bringing about vasodilatation. Allen and McKechnie[13] found no evidence of consistent vasodilatation from the procedure because they did not observe a consistent temperature rise. However, DeTakats, Beck and Roth[6] argue that increased blood flow and vascular capacity are not the same, and that a stretching of the venocapillary bed does not mean a rise in skin temperature. Since 1936, Collens and Wilensky[8] have reported further successes with their apparatus. Kramer[14] and Brown and Arnot[15] also favor the use of this type of therapy. Harpuder and Stein,[16] utilizing the principle of venous hyperemia, obtained good results in the treatment of indolent ulcers. Beardwood, Jr.,[10] does not think it as valuable as suction and pressure. Linton and his coworkers,[17] in an experimental study on animals with the thermostromuhr, concluded that therapeutic venous occlusion increases the arterial blood flow to an extremity. Krusen[18] admits that good results have been reported following the use of intermittent venous occlusion, but states that it is becoming increasingly apparent that there is little if any change in the cutaneous temperatures when any of the merhanical devices are employed. Wright[19] is unconvinced of the value of the method, and feels that it is harmful in certain instances. Abramson et al.,[22] from plethysmographic studies, concluded that this method of treatment did not result in the production of a significant increase in peripheral blood flow.

With these claims and counterclaims it is difficult to decide the efficacy of treatment by intermittent venous occlusion. In our experience, the method is valuable. We have frequently seen patients with excruciating pain due to ischemic neuritis, ulcer, or gangrene reach restful slumber after application of the constricting cuff. Symptomatic relief has been somewhat more consistently obtained by this than by other mechanical devices we have used. Indolent ulcers have healed; gangrene of toes or digits has been demarcated. However, our experience with this modality is admittedly less than with the suction and pressure apparatus. (See table 2.)

TABLE 2. — *Results of Treatment of Peripheral Vascular Diseases With Intermittent Venous Occlusion as the Chief Mechanical Method.*

Diagnosis	Number of Cases Treated	Average Hours of Treatment	Number Improved	Percentage Improved
Arteriosclerosis obliterans	6	63.5	5	83
Arteriosclerosis obliterans with diabetes	3	49.0	2	66
Thromboangiitis obliterans	15	53.7	10	66
Arterial embolism (residuals of)	1	61	0	0

In the employment of intermittent venous occlusion the duration of treatment, the timing of pressure and release, and the degree of pressure are to be varied to meet the demands of the local condition and the reaction

of the individual patient. It is imperative that contraindications be scrupulously regarded. After considerable trial, we have found that it is best to start with a low pressure, usually 30 mm. (mercury), and gradually to increase this to a maximum of 80 mm. The most satisfactory time interval is 1½ minutes of pressure alternated with 1½ minutes of rest. The minimal period of treatment is 1 hour. Many patients are treated for 2 to 4 hours or more, as indications suggest, and in certain instances as much as 24 hours treatment may be indicated. As an adjuvant, we erect a cradle, containing a lamp, at the foot of the patient's bed, so that the environmental temperature may be kept approximately between 96 and 100 F.

The Oscillating Bed

The third major development in the mechanotherapy of peripheral vascular diseases is the oscillating bed. This apparatus reproduces postural exercises without any effort on the part of the patient, and can therefore be carried out for long periods, during sleep, and for debilitated patients. The method was first described by Sanders[20] in 1936. Horton, Krusen, and Sheard[5] think that this is undoubtedly the best mechanical device now in use. Barker,[21] using a modification of the Sanders bed, treated a variety of vascular conditions over a 3-year period, with consistently good results in 58 per cent of the cases. He noted the following physiologic effects of this treatment: Definite rhythmic changes in color of the feet and toes, i. e., pallor on elevations and rubor on dependency, indicating alternate emptying and filling of the capillary bed; (2) rather striking and rapid relief of pain of vascular origin in a large number of cases during the time that the patients were on the beds; (3) the induction of sleep in a large number of cases, even in patients who had had great difficulty in sleeping, due partially to relief of pain; and (4) definite but incomplete vasodilatation, as evidenced by rise in the skin temperature of the digits.

Our results with the oscillation bed have apparently been satisfactory, but not as definitely so as with the other two mechanical devices. We have not used the bed alone in the treatment of any given case of peripheral vascular disease, and cannot, therefore, properly evaluate the results. Fifty per cent of 20 patients who were given this treatment stated, after 3 hours (which is the average duration of the treatment in this clinic), that their legs and feet felt warmer and more "life-like," and that the pain (whether due to ischemic neuritis, ulcer, or gangrene) was lessened. (See table 3.)

TABLE 3. — *Symptomatic Relief With Oscillating Bed in Peripheral Vascular Diseases.*

Diagnosis	Number of Cases Treated	Average Hours of Treatment	Number Relieved	Percentage Relieved
Arteriosclerosis obliterans	10	201.5	5	50
Arteriosclerosis obliterans with diabetes	3	328	2	66
Thromboangiitis oblitegans	7	162.8	3	43

The other patients in this group, either obtained no relief from their pain or noted no subjective change in the temperature of the part. Because of this, we give the patients who are selected for this treatment a few trial periods. If these result in no subjective improvement, other methods of treatment are turned to. The periods of treatment with this device are so lengthy that only a few patients can reasonably be handled in a 24-hour period. We select those patients whose condition seems more severe, and

warrants prolonged postural exercise. And we have erected a cradle at the foot of the bed of the patient, to produce an environmental temperature, over the lower extremities, of about 96 to 100 F. The patient is thus given the advantage of prolonged postural exercises while the peripheral vessels are in a state of vasodilatation within the limits of their altered physiology.

While the three machines described in the foregoing are presently at the forefront in the physical therapy of peripheral vascular diseases, other and more commonly available modalities still have a place in this field. Thus, hydrotherapy remains an important treatment measure. It was formerly our practice to use contrast baths routinely, in the absence of contraindications. But we have largely discarded this practice, because of the danger of adding severe vasospasm to a limb which already is suffering from the ischemia of organic occlusive disease. We might add that we have not observed any ill effects from this practice in our clinic. The gentle massage and vascular relaxation afforded by the warm whirlpool bath, maintained at a temperature of 110 F., are valuable. Wherever feasible, patients with arterial disease are given a 20-minute course of treatment of the affected extremities. The sitz bath, too, is utilized at a similar temperature for same period. This tends to produce reflex vasocillation for at least a short time.

In the presence of gangrene, ulcer, or evidence of inflammation, strict bedrest is enforced within reasonable limits, dependent upon the severity of the case. Patients with no open lesions are permitted activity within the limits of their vascular capacity; that is, they are not allowed to walk to the point of development of symptoms of ischemia. These patients who are restricted to bed present the added problem of muscular atrophy, which is combated by means of massage and limited exercise. Most of our vascular cases have a bed-cradle, with heat controlled between 96 and 100 F., so that whenever they rest and sleep they are exposed to optimal temperature. The patients are instructed as to potural exercises and urged to carry these out at definite periods, so that they will fit into their treatment without conflict.

One of the standard modalities utilized in physical therapy, the galvanic current, has proved of restricted value in the treatment of selected cases of peripheral vascular disease. Mecholyl chloride is introduced into the skin in areas surrounding indolent ulcers, or into extremities with evidence of vasospasm. This drug, which has a parasympatheticomimetic property, produces vasodilatation in the local tissues for a variable length of time. We have utilized this principle of electrophoresis in selected cases, employing a 1:200 concentration of the drug. Our experience with this modality in peripheral vascular diseases has been limited; but, in the few cases we have used it, subjective improvement at least has resulted.

An associated difficulty in the management of peripheral vascular disorders is the complications to which these patients are subject. Those complications range from claudication to ulcers or gangrene. However, the prophylactic care of early cases, and of patients who have recovered from tissue damage or loss with healing, is of equal or greater importance. The development of a fixed program of treatment impresses the patient with the need for constant vigilance. This is particularly important in an individual who has had but mild symptoms, such as numbness of a digit, but who, upon examination, is discovered to have well-marked occlusion of major arterial pathways with an as yet adequate collateral circulation. Whereas such a patient would be disposed to minimize the seriousness of this condition if he were treated haphazardly, a vigorous and regular course of

therapy, in addition to its intrinsic value, tends to educate him as to the importance of prophylactic control. He comes to realize that the healing of an ulcer or the spontaneous separation of a gangrenous digit with resolution of the residual lesion, has not really cured his disease. As a further means of education of the patient in prophylaxis and treatment, he is urged to continue postural exercises upon discharge from the hospital. The necessity for the development of a program of home treatment to such a point that its various phases become routine and habitual, is stressed. The psychologic effect of the intensive treatment in the hospital and following discharge is by no means insignificant. The hopeless attitude of a patient who is afflicted with a peripheral vascular disorder is often replaced by one of encouagement and cooperation. The advancement in the knowledge and intelligent management of these conditions is reflected in the greater confidence of the patient in the physician. It is our opinion that the most rational approach to the problem is a program of intensive treatment, one that combines the modalities best suited to the individual.

The treatment administered to patients in our clinic is varied to meet their needs and conditions. We frequently have occasion to treat them, with two or perhaps all three of the mechanical devices. It is for this reason that our conclusions are colored by the subjectivity of the patient. With sufficient care in extracting information from the patient, the data obtained, while not of scientific accuracy, are still of sufficient value to enable us to arrive at satisfactory conclusions as to the therapeutic merits of the machines. To illustrate the operation of our treatment schedule, the following is an outline of a day's routine for an average case of arteriosclerosis without diabetes, with no open lesions, but with evidences of ischemia in the lower extremities:

7-7:30 a. m.	Postural exercises.
7:30-8 a. m.	Breakfast.
8-9 a. m.	Rest and massage with lanolin or olive oil.
9-9:30 a. m.	Whirlpool bath.
9:30-10 a. m.	Postural exercises.
10-12 noon	Suction and pressure.
12-12:30 p. m.	Lunch.
12:30-1 p. m.	Rest.
1-1:30 p. m.	Postural exercises.
1:30-2 p. m.	Sitz bath.
2-4 p. m.	Intermittent venous occlusion.
4-4:30 p. m.	Rest.
4:30-5 p. m.	Postural exercises.
5-5:30 p. m.	Dinner.
5:30-7:30 p. m.	Rest.
7:30-8 p. m.	Postural exercises.
8-9:30 p. m.	Rest and massage with lanolin or olive oil.
9:30-10 p. m.	Postural exercises.

This schedule is adhered to as closely as possible, dependent upon the cooperation of the patient and the use of other methods of treatment such as typhoid vaccine, etc. In selected cases the duration of treatment with the mechanical devices is shortened or lengthened, as indicated. The patient whose condition warrants the use of the oscillating bed is permitted to sleep on this device all night. Others are chosen for minimal periods of three hours during the day.

Table 4 is a recapitulation of the cases of peripheral vascular disease

TABLE 4. — *Summary of All Cases of Peripheral Vascular Disease Treated by the Three Mechanical Devices.*

		Type of Treatment						Results of Treatment			
	Number of Cases	Os. Bed Cases	Os. Bed Hours	Suc. Press. Cases	Suc. Press. Hours	Int. Ven. Oc. Cases	Int. Ven. Oc. Hours	Unimproved	Improved	Per Cent Unimproved	Per Cent Improved
Arteriosclerosis obliterans	29	10	2,015	19	1,225	6	380	5	24	17.2	82.8
Arteriosclerosis oblit. with diabetes	18	3	985	14	831	3	149	2	16	11.1	88.9
Thromboangiitis obliterans	29	7	1,140	27	1,841	15	806	7	22	24.1	75.9
Frostbite	4	....	.....	4	424	....	.....	0	4	.....	100
Arterial embolism (recent)	1	....	.....	1	21	....	.....	1	0	100	.....
Arterial embolism (residuals of)	3	....	.....	3	175	1	61	1	2	33.3	66.7
Arterial thrombosis (undeter. cause)	2	....	.....	2	115	....	.....	1	1	50	50
Enderarteritis (syphilitic)	1	....	.....	1	60	....	.....	0	1	.....	100
Aneurysm, popliteal (postoperative)	1	....	.....	1	45	....	.....	0	1	.....	100
Polycythemia vera w. (arterial thrombosis)	2	....	.....	2	68	1	45	0	2	.....	100

which were treated with the three mechanical devices described in this report. Of the patients included in this statistical summary, a number subsequently were treated by radical surgery. This does not, however, contradict our claims that they were improved, at least subjectively or temporarily, by these physical methods.

Summary

1. The importance of accurate diagnosis, proper selection of cases for the particular type of treatment, and correlation of physical, medical, and surgical methods of management in peripheral vascular diseases, is stressed.

2. Intermittent venous occlusion has, in our experience, been the most valuable of the three methods of mechanotherapy in allaying symptoms and healing open lesions.

3. The suction and pressure method still plays an important role in this field.

4. A program of intensive therapy, regardless of which modality is used, is important in principle.

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ARCHIVES of PHYSICAL THERAPY
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.·. EDITORIALS .·.

THE TWENTY-SECOND ANNUAL CONGRESS

After reviewing the program which has been prepared for the twenty-second Annual Session of the Congress, your editor is impressed with the timeliness of the subjects to be presented. The dozen essays on rehabilitation of children, industrial workers and disabled service men furnish material which no physician, who practices physical medicine even in a small way, can afford to miss. We all consider it our patriotic duty in this time of national emergency to do our jobs in the most efficient and effective manner. Anything which contributes to our knowledge and increases our usefulness should by no means be neglected. If you are one of those who have decided not to attend the Congress this year, will you not reconsider? Get out your July issue of the ARCHIVES, review the program again, and see if you still think you can afford to miss the Chicago meeting. We shall expect to meet you at the Twenty-second Annual Congress Session.

THE BRITISH ASSOCIATION OF PHYSICAL MEDICINE

In the May-June issue of *The British Journal of Physical Medicine* and *Industrial Hygiene*, there was announced the organization of The British Association of Physical Medicine. In his presidential address before the Section of Physical Medicine of the Royal Society of Medicine last October, Philippe Bauwens indicated the need for some definite program to bring order into this field and made the following recommendations: (a) the awarding of a diploma in physical medicine to physicians practicing it; (b) the formation of a committee of "selected informed practitioners of physical medicine" to study the problem of organization; (c) the development of a "Faculty of Physical Medicine" with a diploma in physical medicine as a requirement for membership and a thesis based on original work as a qualification for fellowship; and (d) eventually a "Chair of Physical Medicine."

After six months of committee work, the new society, The British Association of Physical Medicine, has been born. The editor is sure that he speaks for the officers and membership of The American Congress of Physical Therapy when he extends to our British colleagues our heartiest congratulations and our sincerest good wishes for the immediate and continued success of the new Association. May our two organizations advance together in close cooperation in the further scientific development of physical medicine.

CARBON DIOXIDE AS A THERAPEUTIC AGENT

There are few substances in the medical armamentarium of wider usefulness than carbon dioxide. The reasons for its extensive employment in therapy are the facts that it is relatively harmless and that it exerts important and definite physiologic effects. It is essential to life itself through

its stimulating action on the respiratory center. This is the basis of some of its most important therapeutic uses.

Locally, carbon dioxide produces a prickling sensation of the skin and mucosae, succeeded by mild local anesthesia. There follows vasodilatation and redness. This local effect, together with certain reflex responses, is the rationale of the well known Nauheim bath. Its action on mucous membranes in the form of carbonated water makes it useful as a carminative, as a remedy for nausea, and as a stimulant to gastric secretions. By its carminative action it increases gastric motility and hastens emptying. The dermatologist uses carbon dioxide snow for the distruction of benign lesions of the skin. The pathologist employs it as a freezing agent in the rapid diagnosis so important in cancer surgery.

The central respiratory stimulation produced by carbon dioxide is the underlying reason for another series of important uses. When the concentration of carbon dioxide in inspired air reaches 2 per cent, respiratory stimulation begins, accompanied by an increase in the tidal air and minute volume. With a 4 per cent concentration the depth of respiration is greatly increased and the rate quickened. At 5 per cent, stimulation becomes marked and respiration, labored. With continued rise in concentration, there is increased augmentation of breathing up to 10.4 per cent above which there is depression.

The respiratory stimulation produced by carbon dioxide has been utilized in the depression occurring in poisoning by carbon monoxide, alcohol, morphine and the barbiturates. It has been employed in all forms of asphyxia including asphyxia and atelectasis of the newborn. It has been advocated by some in the prevention and treatment of postoperative atelectasis, embolism and pneumonia, in pertusis, in hiccough, in bronchial asthma, in surgical shock and in obstetric practice to overcome uterine inertia.

Elsewhere in this issue attention is directed by Banyai and Jurgens[1] to the use of carbon dioxide inhalation to control the cough and dyspnea in advanced and far advanced cases of pulmonary tuberculosis. Using a very simple method of administration, these authors observed: (1) easy and effortless expectoration; (2) diminution of cough; and (3) relief from dyspnea. The subjective responses of their patients were gratifying. The concentration of the gas used was 10 per cent in 90 per cent oxygen. Anticipating the obvious objection to respiratory stimulation in this disease, the authors maintain that it is their belief that the clinical results outweigh the disadvantages and dangers.

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A NEW DEMAND FOR THE SERVICES OF PHYSICAL THERAPY TECHNICIANS

In the July, 1943 issue of the *National Foundation News,* published by the National Foundation for Infantile Paralysis, Incorporated, there appears a report which points out that the number of cases of poliomyelitis is now at its highest peak since 1934.

One portion of this report is of special interest to everyone interested in physical therapy. It is stated that "one of the most difficult phases of providing care for patients in an infantile paralysis outbreak is that of supplying sufficient physical therapists trained in the Kenny method of treat-

ment of this disease. There are less than 2,000 registered physical therapists in the United States. Only a small part of those remaining for civilian practice have had any training in this newer method of treatment of early cases of infantile paralysis. The shift in emphasis in treatment from one of rigid immobilization to one of early physical therapy has created a new demand for the services of physical therapy technicians. That demand cannot be met without careful planning and some sacrifice.

"The National Foundation is doing all it can to supply these trained workers to epidemic areas. Technicians are being borrowed from non-epidemic areas and assigned to hospitals where the need is greatest. It must be realized that there are not enough trained technicians to go around if and when epidemics spread and increase beyond a certain point. A physical therapy technician can care for only a limited number of patients—certainly never more than fifteen or twenty mild cases—and her full time may be taken up by as few as six cases with extensive involvement. Neither nurse nor physician can substitute for the physical therapy technician. To add further to the problem, it is only through special study that the technician can master the details of the early physical therapy of infantile paralysis."

It is obvious that in addition to the increased demand for physical therapy technicians imposed by the war rehabilitation effort, there is also going to be an increasing demand for well trained technicians to give the modern treatment for poliomyelitis. This is just one more reason why our schools for physical therapy technicians must continue unrelenting efforts to graduate as many students as possible and to give them the modern principles of physical treatment including such procedures as the Kenny treatment of infantile paralysis.

Control of Peripheral Circulation — Harpuder

(Continued from page 486)

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MEDICAL NEWS

Lieut. Col. Titus' Rehabilitation Program

If the recording of an interesting bit of news could ever be directed to sources where its recognition is compensated by reward, this item, concerning the creative efforts of our own Lieut. Col. Norman E. Titus would read as a citation and an accolade for services rendered to our Army manpower. The intent of this item while not as ambitious is in effect more satisfying, because it calls attention of our readers to the sacrificing and patriotic labors of a seasoned member and leader in physical therapy, who twice in a busy life is serving his country with a zeal regardless of cost. Demobilization in World War I brought the then Captain Titus back into our ranks with as rich an experience in concerning methods and therapeutic means as any that had been privileged to practice the then limited and somewhat questioned specialty of physical therapy. Mobilization for our present global conflict found Titus in the forefront with others in our organization, seeking assignment without preferment to serve his country as an authoritative and recognized specialist in the care of our Army forces. With spirit courageous, though vision faulty, he has brought to his new duties an elan and a wide experience that has justified his commission of a Lieutenant Colonel in his present administrative position at Lawson General Hospital in Atlanta.

The program developed by Col. Titus concerns the rehabilitation and conservation of Army manpower from the earliest and initial stages of trauma and the like. The objectives are to shorten the time of convalescence and to return patients to military duty in the best possible physical and mental condition. The program violates many so-called accepted beliefs and past traditions of the profession, which have been discarded on the basis of experience and experimental evidence. According to Col. Titus, the patient is not permitted to languish in his own beliefs, phobias or neurotic emotionalism. The intention is to obviate boredom and to keep mind and body active and occupied. The slogan is "never a dull moment," and no one goes AWOL under the new program.

A visitor at Lawson General Hospital sees stalwart, sun-burned soldiers, some in splints and casts, some with a history of a record removal of casts and a shortening of the convalescence; others are seen grouped and directed toward creative play or exercise, in or out of bed. Nothing and no one is permitted to languish or deteriorate. Mind and body, spirit and physique are under special and directive scrutiny and this under an overall program that includes the use of medicine, surgery and physical therapy. This rehabilitation project has as its objective to quicken healing, to strengthen the morale, to orient the handicapped into other useful channels of service, to replace and add to our manpower a residue of servants for service which heretofore became or were designated as the tragic flotsam of military experience.

Activities of Council on Physical Therapy

The Council has devoted much of its time to presentation of useful, reliable and factual information about physical therapeutic methods for the attention of civilian, army and navy physicians confronted with the task of rehabilitation. The Council has cooperated with government agencies by giving advice on physical therapeutic procedures and apparatus. In cooperation with a subcommittee of the National Research Council, the Council has prepared a manual on physical therapy and with other groups is preparing one on occupational therapy, which will be of value to those concerned in rehabilitation. A Handbook of Amputations has also been issued and is of value to both military and civilian physicians. The Council consultants aided in revising the curriculum for schools of technicians and in investigation of audiometers and hearing aids and has published specifications for a home made respirator. Application of ultraviolet radiation for disinfection was studied, and a statement on the problem was published. The booklet "Apparatus Accepted" was revised. Grants were made for research on problems vital to physical therapy. Twenty-one reports were published during 1942.

Raymond Allen Named Dean of Illinois

Dr. Raymond B. Allen, executive dean of the Chicago Colleges of the University of Illinois, has been appointed dean of the medical school, effective September 1. He succeeds Dr. David J. Davis, who is retiring. Dr. Allen will continue in his present position of executive dean of the Chicago departments, which include the colleges of medicine, dentistry and pharmacy and other institutions associated with these units. Dr. Allen graduated at the University of Minnesota Medical School, Minneapolis, in 1928. From 1934 to 1936 he was associate dean in charge of graduate studies at the College of Physicians and Surgeons of Columbia University and associate director of the New York Post-Graduate Medical School and Hospital at Columbia from 1933 to 1936, when he became dean of Wayne University College of Medicine, Detroit. In 1939 he came to the University of Illinois.

Formation of Council on Medical Service and Public Relations

The following abstract covers the report of the Reference Committee on Legislation and Public Relations at the recent meeting of the House of Delegates of the American Medical Association:

There was recommended the formation of a Council on Medical Service and Public Relations to be composed of six members of the American Medical Association geographically distributed over the United States, with the President, the immediate Past President, the Secretary and a member of the Board of Trustees of the American Medical Association:

(*a*) To make available facts, data and medical opinions with respect to timely and adequate rendition of medical care to the American people; (*b*) to inform the constituent associations and component societies of proposed changes affecting medical care in the nation; (*c*) to inform constituent associations and component societies regarding the activities of the Council; (*d*) to investigate matters pertaining to the economic, social and similar aspects of medical care for all the people; (*e*) to study and suggest means for the distribution of medical services to the public consistent with the principles adopted by the House of Delegates, and (*f*) to develop and assist committees on medical service and public relations originating within the constituent associations and component societies of the American Medical Association.

In the exercise of its functions this Council, with the cooperation of the Board of Trustees, shall utilize the functions and personnel of the Bureau of Legal Medicine and Legislation, the Bureau of Medical Economics and the Department of Public Relations in the headquarters office.

Stipulations

Agreements Between Federal Trade Commission and Promoters of Various Products

The following items are abstracts of stipulations in which promoters of "patent medicines," medical devices and cosmetics have cooperated with the Federal Trade Commission to the extent of agreeing to discontinue certain misrepresentations in their advertising. These stipulations differ from the "Cease and Desist Orders" of the Commission in that such orders definitely direct the discontinuance of misrepresentations. The abstracts that follow are presented primarily to illustrate the effects of the provisions of the Wheeler-Lea Amendment to the Federal Trade Commission Act on the promotion of such products.

Bathritis

In October, 1942, one James P. Galligan, trading as Bathritis Company, Chicago, stipulated with the Federal Trade Commission that he would discontinue the following misrepresentations in his advertising of his product: that when used as directed in the bath it would have any therapeutic effect in the treatment of rheumatism, neuralgia or other ailments, or when applied as a liniment would be effective in excess of relieving pain; that it relieves sprains, stiffness and swelling or works toward restoring normal active muscular health; that it is effective in comforting inflamed tissues and dispelling body odors or is a new discovery. — (Reprinted with permission, J. A. M. A.).

Institute for Hospital Administrators

The eleventh Chicago Institute for Hospital Administrators will be held at the Knickerbocker Hotel, August 30-September 10, under the auspices of the American Hospital Association and in cooperation with the American College of Hospital Administrators, American College of Surgeons, American Medical Association and the Chicago Hospital Council. The institute will be under the direction of Dr. Malcolm T. MacEachern, Chicago, and lectures, demonstrations, and round tables will be based on practical means to help solve present-day hospital problems. A minimum of fifty registrants must be assured to hold the institute. Additional information may be obtained from Miss Agnes M. McCann, secretary for the institute for hospital administrators, American Hospital Association, 18 East Division Street, Chicago.

Dr. Victor Johnson Assumes New Duties

Dr. Victor Johnson, associate professor of physiology and dean of students in the division of biological sciences, University of Chicago, on July 1 became Secretary of the Council on Medical Education and Hospitals of the American Medical Association.

Women Physicians in the Army

Dr. Margaret D. Craighill, Dean, has been commissioned Major in the Medical Corps of the U. S. Army. Dr. Craighill is the first woman physician receiving such an appointment. The Army decided recently to extend this opportunity to women physicians. Dr. Craighill will serve in the Division of Preventive Medicine of the Surgeon General's office in Washington, D. C.

Dr. Frances Hellebrandt on Wisconsin State Medical Society Program

Dr. Frances A. Hellebrandt, Associate Professor of Physical Medicine, University of Wisconsin Medical School, will present "Physical Therapy Department for Convalescents in General Hospitals" Tuesday September 14, as part of the round table luncheons being held in connection with the annual meeting of the State Medical Society of Wisconsin, at Milwaukee, September 13 to 15, 1943.

Expand Program of Physical Education

Harold T. Friermood, Dayton, Ohio, has been appointed special secretary of health and physical education of the National Council of the Young Men's Christian Association of the United States of America. He will aid in redirecting the group's program in relation to physical fitness, health education, swimming and water safety and in devising methods of enlisting volunteer and part time leadership for service in communities where staffs have been depleted by transfer of Y. M. C. A. secretaries and physical directors to other forms of service.

The Poliomyelitis Situation

The U. S. Public Health Service reported on July 28 that the incidence of infantile paralysis had reached epidemic proportions in several Southwestern states and that figures for the country during the first six and a half months were the highest in twelve years. The New York Times reported on July 29 that a sharp rise in the number of cases since early June was accounted for by outbreaks in Texas, California and Oklahoma. No specific cause could be assigned for the situation, it was stated, and no predictions could be made as to the possibilities of a further spread. For the first twenty-eight weeks of this year 1,626 cases were listed for the entire country.

Correction

In the publication in the June, 1943, issue of the ARCHIVES of the article by Dr. Simon Stone, on "Artificial Fever and Vitamin Therapy in Treatment for Anterior Poliomyelitis" the footnote should have read: "From the Neurological and Artificial Fever Therapy Services, Elliott Hospital, Manchester, N. H., and the Crippled Children's Services of the State of New Hampshire."

Captain Zankel at Borden General Hospital

A recent issue of the *Borden News* published for the personnel of the Borden General Hospital at Chickasha, Okla., contains an interesting illustrated account of the section of physical therapy, its activities and personnel of which Captain Harry T. Zankel is in charge. Called "reconditioning," the article states "Taking charge of patients in the postoperative period, as well as those with ailments amenable to this type of therapy, the department helps to improve the patient's condition and to repair many of his physical derangements. A subsidiary department is equipped with two fever therapy cabinets for the management of arthritis, certain venereal diseases and other conditions. Two other subsidiary departments are the reconditioning and occupational therapy divisions. With an average of 100 patients each day not including four classes of general reconditioning exercises the physical therapy section is staffed by two commissioned physical therapy aides and eight enlisted men."

Major Bilik at Oliver General Hospital

In the July issue of the ARCHIVES, the name of Major Samuel E. Bilik was inadvertently omitted as chief of the section on physical therapy on the staff of the Oliver General Hospital, Augusta, Ga.

Frank E. Hill 1862-1943

We regret to announce that Dr. Frank E. Hill, of Muncie, Indiana, for many years a member of the Congress has passed away. He graduated in 1890 from the Medical College of Cincinnati and was a fellow of the American Medical Association. We extend our sympathy to his family.

Merrill J. Dorcas

Merrill J. Dorcas, former college instructor and research worker for the National Carbon Company for the past 18 years, died recently at his home in Berea, Ohio. He was 44. Born in Albernett, Iowa, Dr. Dorcas was graduated from Baker University and received his Ph.D. degree in chemistry at Harvard University. After graduation from Harvard he joined the faculty of Wesleyan College in Middletown, Conn., as an instructor in chemistry. Then he was engaged in chemical research at the Bermuda Station of the Rockefeller Institute for Medical Research. He was known to many of the members of the American Congress of Physical Therapy and had participated on many occasions in our programs. We are shocked to learn of his death and wish to extend our sympathy to his family.

Died While in Military Service

Word has been received that a member of the Congress, Robert Titus Phillips of Portland, Maine, died June 11, while a prisoner at a Japanese prison camp in the Philippine Islands. Graduated from Tufts College Medical School, Boston, 1932, Dr. Phillips was a member of the Maine Medical Association, American Rheumatism Association and the International League for the Control of Rheumatism. Dr. Phillips was promoted to Major while a prisoner of the Japanese. Our sympathy is extended to his family.

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BOOK REVIEWS

THE PRINCIPLES AND PRACTICE OF WAR SURGERY. WITH REFERENCE TO THE BIOLOGICAL METHOD OF TREATMENT OF WAR WOUNDS AND FRACTURES. By *J. Trueta*, M.D., Formerly Director of Surgery, General Hospital of Catalonia, University of Barcelona; Assistant Surgeon (E.M.S.), Wingfield-Morris Orthopedic Hospital, Oxford; Acting Surgeon-in-Charge, Accident Service, Radcliffe Infirmary. Cloth. Pp. 425, 144 Illustrations. Price, $6.50. St. Louis: The C. V. Mosby Co., 1943.

In this book, Dr. Trueta has tried to give all of the details of his technic of the treatment of war wounds along with the scientific basis of the methods employed. He has greatly enlarged the scope of the book over his earlier one "Treatment of War Wounds and Fractures" (Hamish Hamilton Medical Books, London; Paul B. Hoeber, Inc., New York.)

He divides the book into two parts—I, Pathology of War Wounds and General Care of the Wounded and II, Essentials of War Surgery. In Part I, he reviews the physiologic knowledge with its application in the treatment of war wounds. He recognizes the defense mechanisms of the body such as control of bleeding and resistance to infection and expects the surgeon to assist Nature in her work of healing. The history of war surgery is outlined carefully to give an understanding of the reasons for his meticulous attention to detail. He gives special credit to Dr. Winnett Orr's fundamental principles—"adequate primary drainage; protection of the patient's wound against secondary infection; immobilization in correct position; and good general care." He claims no new discovery but has applied knowledge in a precise manner to obtain recovery.

He describes carefully the process of wound healing, discussing the effect of projectiles on various tissues and the regenerative processes which follow. Infection is the chief problem in the treatment of war wounds. It would seem that Bernard discussed with Pasteur the relative parts played in an infection by bacteria on the one hand and by living tissues on the other. Pasteur attacked the idea. Now it is recognized that one animal or tissue is susceptible while another is not. The germ is the same; the variable factor lies in the receptor medium. Pyogenic infections, gas gangrene and tetanus are discussed individually and in detail. The physiology, diagnosis and treatment of shock is described. Blood transfusion as a part of surgical treatment of war casualties is a development of the Spanish War which is now universally accepted. The organization of a system by which patients may be transferred soon after injury is given. The problems of anaesthesia in war time are considered from the viewpoint of the surgeon rather than the anaesthetist as it is an expression of Dr. Trueta's experiences.

Part II, "Essentials of War Surgery" takes up the specific treatment of wounds. The biological treatment of wounds rests on five basic principles, every one of which must be applied to ensure the greatest possible success in the treatment of recently inflicted wounds.

1. Prompt surgical treatment.
2. Cleansing of the wound.
3. Excision of the wound.
4. Provision of drainage.
5. Immobilization in a plaster-of-paris cast.

All points are vital, but their successful application turns on Point 3, excision, and excision is closely linked and to largely on Point 1, early surgical treatment, as a period of six to eight hours may permit the contaminated area to become so extensive as to make excision a correspondingly serious operation. Point 5, immobilization in a plaster cast also depends on Point 3, for unless excision is complete the treatment of a recent wound in a closed plaster is fraught with danger. Sutures, skin grafts, articular wounds, amputation, regional surgery, and burns are discussed individually.

The book is of value to anyone having anything to do with traumatic conditions either in civilian or military practice.

PICTORIAL HANDBOOK OF FRACTURE TREATMENT. By *Edward L. Compere*, M.D., F.A.C.S., Associate Professor of Surgery, Northwestern University Medical School; Chairman, Department of Orthopaedic Surgery, Wesley Memorial Hospital; Consulting Orthopaedic Surgeon, Chicago Memorial Hospital; and *Sam W. Banks*, M.D., Associate in Surgery, Northwestern University Medical School; Attending Orthopaedic Surgeon, Chicago Memorial Hospital. Cloth. Pp. 351. Price, $4.25. Chicago: The Year Book Publishers, Inc., 1943.

Compere and Banks have prepared a beautifully illustrated textbook on fracture treatment which will be of extreme value to any physician who is desirous of a rapid "brush-up" on the subject. The book contains no less than one hundred and seventy-one illustrations. Most of these are excellent line drawings which are readily understandable. The authors have prepared this book especially for "the doctor in general practice" and for "the medical student, intern or hospital resident." Their intention was "to provide the mental or visual picture of the actual steps required to reduce, immobilize and provide intelligent after-care for each of the more common fractures or dislocation injuries." They have been successful in achieving their purpose. Despite the numerous texts on treatment of fractures there is probably no small book which covers the subject so succinctly and yet for the most part so completely.

It is unfortunate that, in their efforts to be concise, the authors have devoted only two and a half pages to the "after-treatment of fractures"—one who is interested in physical therapy will regret that so little attention was paid to this important phase of fracture management. Also in view of the fact that the Roger Anderson splint and the Stader splint are now being employed extensively in military surgery it is unfortunate that practically no discussion of these is presented. But perhaps the authors rightly assumed that these complicated splints were not for the audience which they have chosen. Any physician who is desirous of obtaining a concise, well illustrated and intelligible manual of treatment of fractures will do well to consider this handbook.

LAUGH AT THE LAWYER WHO CROSS-EXAMINES YOU. A COURT ROOM ANTIDOTE. By *Charles I. Cusamano,* LL.B., of the New York Bar. Cloth. Pp. 362. Price, $3.00. New York: Old Faithful Publishing Co., 1943.

This book, although written in a light, amusing fashion, deals seriously with the problems of being a witness in a court of law. Under subtitles such as "A Jolly Jury or a Dozen Dead Fish," or "A Pair Always Beats an Ace," sound suggestions for replying to questions while on the witness stand are given. The author believes that most persons who are called as witnesses go to any length to avoid such duties. Those that do appear are generally so nervous that their testimony is not particularly valuable and they themselves feel dissatisfied with the experience. The author further believes that witnesses could derive a great deal of individual satisfaction as well as perform a greater service to society when they appear in court, if they would follow certain suggestions he has endeavored to make. Since this entire book of fair size is devoted to advice and instruction in regard to the conduct of witnesses, it can be seen that the author goes into considerable detail. Some of the chapters are devoted to these rules: Preserve the evidence, cooperate with the lawyer for your side, avoid contradictions before the trial, review your facts before you testify, understand the theory of your side, aim to convince, but do not memorize and be ready for cross-examination. The book as a whole will no doubt be of interest to members of the medical profession, but they will be especially interested in the chapter "Fruit Fit for Experts" and in particular the section entitled "Doctors." The author states, "The expert witness who is most frequently in court is the doctor. He has to act, as it can be seen, in two capacities. First he attends the patient and then he must come to court to testify. His direct testimony generally is given in the following order: 1. Qualifications. 2. Condition of patient, services rendered and other related facts. 3. Opinions. 4. Value of his services. The author enlarges on his subject and presents facts and information under subtitles such as "Padding Medical Bills Brings Headaches and Ills," "Doctors Who Testify for Plaintiffs Cover Much More Ground Than Medicos Who Testify for the Defendants" and "The Doctors' Prime Hypothetical Question Is the One Which Shows the Causal Relation." The book is written from a carefully worked out outline which makes it well organized. It is written in simple language with the use of a minimum of legal terms. It can be recommended as interesting and valuable reading for the physician who might be called to testify in court or to anyone who would enjoy an understanding picture of court proceedings relative to a witness, expert or otherwise.

DOCTOR IN THE MAKING. THE ART OF BEING A MEDICAL STUDENT. By *Arthur W. Ham,* M.B., Associate Professor of Anatomy, in Charge of Histology, Faculty of Medicine, University of Toronto; Honorary Secretary of the Banting Research Foundation; and *M. D. Salter,* M.A., Ph.D., Lecturer and Research fellow in the Department of Psychology, Faculty of Medicine, University of Toronto. Illustrations by *Jean McConnell.* Cloth. Pp. 180. Price, $2.00. Philadelphia: J. B. Lippincott Co., 1943.

Although this book is designed primarily for medical students and for those who contemplate becoming medical students, much of the material that is presented would apply to students in other college and university courses and indeed to the progress of people in many walks of life. The authors in their preface explain that most of their material was gathered from their experiences as members of an advisory committee for students in their first year of the medical course. They found many discrepancies between prediction and accomplishment as based on aptitude tests and past scholastic records. They therefore concluded that ability and academic preparation do not guarantee success. Habits of study, attitudes, general personality characteristics and other factors were found to be of considerable importance in determining whether a student with good academic preparation and mental equipment would achieve the success predicted for him. While it is written in popular style with humorous illustrations, and therefore highly entertaining nevertheless it is a good presentation of the problem of how to lead the student to the "right" road to learning.

NUTRITION. By *Fern Silver,* Supervisor of Home Economics, Lincoln High School, Albuquerque, New Mexico; Author of "Foods and Nutrition." Cloth. Pp. 168. Illustrated. Price, $1.35. New York and London: D. Appleton-Century Company, 1943.

Mrs. Silver says: "One cannot believe that the great extent of malnutrition that exists among the population of this land as revealed by an abundance of statistical material is caused altogether by the inability of the homes to purchase food adequate for family needs. Much of it must be attributed to a poor knowledge of nutrition requirements." The physician can recommend this book for his patients especially for the mother in the home. It is a concise, easily understood factual discussion of the relation between foods and bodily energy and

growth in which is explained the significance of calories, carbohydrates, fats, proteins, minerals and vitamins. It skillfully condenses the findings of the latest scientific research in the field of nutrition in a brief, simply written exposition and a number of graphic charts that are within the grasp of every person.

VITAMIN VALUES OF FOODS. A COMPILATION. By *Lela E. Booher,* Formerly Senior Nutrition Chemist, Now Director of the Institute of Nutrition, Milwaukee Children's Hospital, Milwaukee; *Eva R. Hortzler,* Formerly Assistant Chemist, Now with Bio-Chemical Research Laboratories, Parke, Davis & Company; and *Elizabeth M. Hewston,* Associate Chemist, Bureau of Home Economics. Reproduction of No. 638 produced by Department of Agriculture, 1942. Cloth. Pp. 224. Price, $2.75. Brooklyn: Chemical Publishing Co., Inc., 1943.

There is presented a summary of the vitamin A, thiamin, ascorbic acid, vitamin D, and ribaflavin values of foods in terms of International Units or absolute weights of these vitamins, as recorded in the literature through December, 1940. Attention is directed to the list of references to the literature on the subject as stated through December, 1940. An authentic reference book of this type is important in these days of the popularity of vitamins.

HOW TO KEEP FIT AND LIKE IT. A MANUAL FOR CIVILIANS AND A PLAN FOR A COMMUNITY APPROACH TO PHYSICAL FITNESS. By *Arthur H. Steinhaus,* Ph.D., M.P.E., Professor of Physiology, George Williams College; *Alma M. Hawkins,* A.M., Assistant Professor of Hygiene and Physical Education, George Williams College; Lt. Comdr. *Charles D. Giauque,* U. S. N. R., Formerly Professor of Health and Physical Education, George Williams College; and *Edward C. Thomas,* B.S., Physical Director, South Chicago Y. M. C. A. Illustrated by *Lea Waterman.* Paper. Pp. 64. Price, 25 cents. Chicago: Basic Books, Inc., 1943.

This is one of the sixteen booklets in "Nothing Counts but Victory" Series. In pamphlet style it contains exercises and programs to be used for individual or group instruction, how to plan and organize fitness classes, how to select and train instructors and how to test and score the physical ability of the person. Much of the material is now being used for training men and women in the service.

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PHYSICAL THERAPY ABSTRACTS

Tuberculous Tracheobronchitis. Louis H. Clerf.

Dis. Chest **8**:356 (Nov.) 1942.

There appears to be considerable difference of opinion regarding the plan of treatment to be employed endobronchially in tuberculous tracheobronchitis. This is in great part due to the recent acquisition of our knowledge concerning this condition and also that inadequate time has elapsed to permit arriving at final conclusions. There still is difference of opinion regarding the treatment of tuberculous laryngitis. There are those who believe that treatment of infiltrating and ulcerogranulomatous lesions is of no avail since the disease either will progress or recover spontaneously without endobronchial manipulations. Irradiation therapy has been recommended with variable results. Kernan has employed ultraviolet lamp therapy to the nonulcerative, infiltrating lesions and is of the opinion that it exerts a beneficial effect.

Ulcers are commonly treated by some form of cauterization.

March Fractures. Howard E. Sweet, and Wendell H. Kisner.

J. Bone & Joint Surg. **25**:188 (Jan.) 1943.

The author's treatment for this not uncommon condition is immobilization of the foot in a plaster boot for a period of four to eight weeks, depending on the roentgenographic findings of adequate callus formation. Following removal of the plaster boot, the patient is given one to two weeks of rest and physical therapy and then is returned to full military duty. In several of these cases, it has been necessary to readmit the patient to the hospital for an additional week of rest and physical therapy, because of continued pain in the foot on weight-bearing.

In conclusion, it should be pointed out that the incidence of march fracture is increasing, because of the rapid growth of the army, and the making of a correct diagnosis is easy when this condition is kept in mind.

The Effective Stimulus for Increased Pulmonary Ventilation During Muscular Exertion. Julio M. Barman; Manoel F. Moreira, and Frank Consolazio.

J. Clin. Investigation **22**:53 (Jan.) 1943.

The authors summarize their work as follows: 1. The increase in pulmonary ventilation has been studied in normal men exercising the limbs, first with normal circulation, and then with the circulation cut off by pressure from inflated cuffs. 2. Light exercise (at a rate of 12 kilogrammeters per minute) of the flexors of the hand during total ischemia of the arms usually resulted in a smaller ventilation than when the arms had normal circulation. Release of the ischemia was followed at once by a marked hyperventilation. 3. In subjects walking uphill on a treadmill (at a rate of 700 kilogrammeters per minute for a 90 kilo subject), total ischemia of both legs resulted in a marked diminution of pulmonary ventilation, even though the nervous pathways were intact, and release of the ischemia was followed at once by a marked hyperventilation, much higher than the value prior to ischemia. 4. These results are interpreted to mean that the chemical stimulus for increased ventilation in exercise of these types is far more important than the reflex.

Problems of Neuroses in Industry. Ernest H. Capel.

Practitioner **169**:354 (Dec.) 1942.

The treatment of accident neuroses should begin the moment an accident has been sustained, which is much the same as saying that treatment should be preventive. An accident is a psychologic shock besides being a physical one and unfortunately thoughtless handling and tactless remarks made at the time of the accident frequently increase the shock. The attitude of the company to its employee forms perhaps the most important part of the background on which the accident is staged. That the employee should have confidence in his firm, know that he will get consideration, a fair deal and a secure future, is of the utmost importance. The tactless handling of patients by lawyers and insurance agents frequently antagonizes them and makes them fear the worst. Solicitors, anxious relations and practitioners interested from the patient's own viewpoint, often do irreparable harm by exaggerating the possible consequences and suggesting possible ulterior motives on the part of the employer. The patient's own family practitioner is frequently to blame in exaggerating the seriousness of the accident, partly to be covered if anything goes wrong and possibly to gain more kudos if all goes well. It is essential that a physical diagnosis and prognosis be made as early and as accurately as possible, and this should be conveyed exactly to the patient. There should be a strong suggestion of positive recovery and no mention of after-effects, except those that will reasonably be expected. In the case of minor injuries, immediate return to the same work whenever possible is desirable. In other cases rehabilitation on suitable work should be started as soon as possible. A few words of caution are necessary here. Although it is desirable to get a patient back to work as soon as possible, the return to a job that is "made" for the purpose, at which he is obviously not earning

his pay and which he himself can see will never lead him back tc his old work, will do more harm than good. It will merely serve to fix in his mind what he already fears, i.e., that he will never get back to his old job or earn full money again. Rehabilitation must lead a man step by step back to doing his old job. The steps must be explained to him and he must understand that he is himself responsible for recovery and getting from one step to the next. It is not always possible to arrange alternative work which will at the same time provide rehabilitation. In this case it is important that rehabilitation in some other form should be arranged and carried on, even though he continues to work.

The Levator Ani, Coccygeus and Piriformis Muscles. Agents in the Causation of Coccygodynia, Superior Gluteal Pain and Sciatic Syndrome. Thomas Wilensky.

Am. J. Surg. **59**:44 (Jan.) 1943.

Massage was originally used to discover whether or not the digitally diagnosed muscle spasm could be relieved by this simple method. In the event that the patient does not show a definite degree of subjective improvement after the first four to six massages over a period of approximately two weeks, orthopedic or other consultation should be sought.

Thiele's series had an average of eleven treatments over an average period of eleven weeks.

Wilson, discussing Thiele's paper, pointed out the interesting observation that eight out of twenty-two patients in his series complained of pain in the lower portion of the abdomen. In every instance this pain followed the course of the iliohypogastric and ilioinguinal nerves and could be exaggerated by pressure on the piriformis muscle near its origin on the affected side. Six of this group of eight cases were relieved by massage of the piriformis muscle alone and the other two were cured by a combination of anal operation and massage.

Obviously there remains a great deal to be done and said concerning this general problem of low back pain, coccygodynia and sciatic syndrome. It has not been the intention of the writer, in this discussion, to give the impression that the described conditions and the indicated nerve-musculo-fascial relationships constitute definite clinical entities for which satisfactory treatment can be invariably delivered.

On the contrary it is the purpose of the paper to emphasize the loose linkage of cause and effect as indicated for these states and to point out that so much of the evidence may be classed as circumstantial.

Nevertheless it is the author's conviction that the muscles and related structures can serve as the direct or indirect causative agents in various cases of low back and sciatic syndrome pain. Therefore, all such states have not been adequately studied unelss this consideration has been explored.

Postural Changes in Respiration. Elizabeth Brogdon Franseen, and F. A. Hellebrandt.

Am. J. Physiol. **138**:364 (Jan. 1) 1943.

Respiratory movements are sometimes considered as a subsidiary line of defense against orthostatic collapse by virtue of expiratory compression of the abdomen and inspiratory thoracic suction. Hill and Barnard in 1897 showed that when splanchnic vasomotor tone of their animals was destroyed, respiratory efforts maintained the circulation, though far less efficiently. On the basis of plethysmographic experiments with dogs in 1933, Eyster and Hicks believe that the influence of the extent of breathing on venous return is not as great as is ordinarily stated. More recntly, Boyd and Patras (1941), using a cardiometer in the closed chest, have reported increase in diastolic and stroke volume with inspiration which were exaggerated by the deep and prolonged breathing following vagotomy.

In the authors' experiments it is impossible to evaluate the importance of this respiratory pump as an aid to the circulation. Respiratory behavior is shown to be closely related to cardiovascular competence. It is augmented as the gravitational handicap to the circulation, is made greater and approaches normal when the compensatory action of postural sway reduces that handicap. With few exceptions both fainter and non-fainters responded with some augmentation of ventilation to protracted support in near-verticality. However, the point of failure of the circulation did not appear to bear any time relationship to the percentage increase in ventilation. Whether the increase in breathing prolongs the period of tolerance for the erect position through aspiratory action can only be a matter of conjecture. The fact that the increases observed were in many cases due to rises in rate with reductions in amplitude renders this unlikely. The augmented ventilation seems rather a reflection of the general slowing of the circulation in which the medullary centers are more or less seriously deprived of oxygen unless more effective compensatory mechanisms combat the pull of gravity on the blood column. Of these, the rhythmic squeezing action of muscle is clearly important.

The authors conclude that respiratory changes induced by alteration of posture outstrip concomitant metabolic increases as verticality is approached. Increases in ventilation in vertical postures appear to be secondary to interference with blood flow to the head. Postural sway is a factor of material benefit in combatting orthostatic hyperpnea.

The Results of Poliomyelitis in Baltimore. Raymond E. Lenhard.

J. Bone & Joint Surg. **25**:132 (Jan.) 1943.

The report of the results of poliomyelitis in Baltimore is a study of the epidemic of 1941, with particular reference to the recovery of persons and of extremities. In the report is also a study of cases of poliomyelitis treated in previ-

ous years and observed for the degree and the rapidity of recovery of specified muscles.

In order to record the results in the after-care of poliomyelitis, it seems necessary to have some measurement of the degree of muscle involvement. Until some better method of estimating muscle power is devised, it is possible to isolate muscles and to express their action in terms of strength, which can be graded in percentages of normal. If a well trained physical therapist examines the same patient throughout the duration of the treatment, the record is an accurate estimate of the progress of the paralyzed muscles.

These studies offer a basis for the comparison of epidemics. In 1941 in Maryland 68 per cent of the patients recovered and 14 per cent more had a slight residual weakness. Only 2 per cent had complete disability. The treatment was protective care and physical therapy for weak muscles. There was no complete immobilization of the patient or of extremities.

The patients who do not recover rapidly or spontaneously need prolonged treatment in an effort to bring the weak muscles to their maximum power.

Muscles do not improve in direct ratio to the degree of initial weakness and may continue to improve for eighteen months in those cases treated immediately after the onset of the poliomyelitis. Patients seen later may improve for a period up to nine months.

Combined Artificial Fever-Chemotherapy in the Treatment of Neurosyphilis. H. Worley Kendell, Walter M. Simpson, and Donald L. Rose.

Brit. J. Phys. Med. **6**:15 (Jan.-Feb.) 1943.

After a decade of rich and constantly enlarging experience with physically-induced artificial fever therapy, the successful results achieved by many workers in many lands are sufficiently comparable to justify the categorical statement that artificial fever therapy is at least as effective as malaria therapy in the management of symptomatic or asymptomatic neurosyphilis. In the hands of many reliable investigators artificial fever therapy has yielded significantly superior results. Patients who have not responded well to malaria therapy have been benefited by subsequent artificial fever therapy.

There is unmistakable evidence that the extent of clinical improvement in neurosyphilitic patients is related directly to the height and the duration of fever. The thoroughly documented analyses made by statisticians of the U. S. Public Health Service provide proof that the clinical responses were distinctly inferior when relatively low temperature levels were employed. There is no longer any valid reason to assume that the malaria plasmodium exerts any specific effect upon *Spirochaeta pallida*.

Several investigators who have had long experience with malaria therapy and with artificial fever therapy have abandoned malaria therapy in favor of artificial fever therapy. This decision was determined largely by the observations that patients tolerate artificial fever therapy more readily, that a high proportion of neurosyphilitic patients can be treated with artificial fever on an ambulatory basis without interruption of gainful employment, and that treatment complications are minimized with artificial fever therapy.

Since continuous hospital treatment is not a requirement for the artificial fever treatment of most neurosyphilitic patients, the cost of the treatment is not necessarily greater than with malaria therapy. If the patient remains at his regular employment during the course of artificial fever treatments, the cost is usually much less.

The employment of auxiliary chemotherapy in conjunction with any type of fever therapy is an essential requirement. There is evidence that the concurrent use of chemotherapy with artificial fever therapy increases the therapeutic effectiveness of both agencies.

The hazards inherent in the various physical modalities used for the production of artificial fever during the first few years of the undertaking have been largely eliminated. Recognition of the physiologic changes produced by fever has led to the utilization of means to combat such effects. A procedure which was looked on as a technical feat a decade ago may now be applied by skilled workers with a frequency which approaches routine.

Developments of the past few years have demonstrated that equally favorable clinical and serologic results may be obtained with fewer total hours of artificial fever therapy, applied in shorter individual sessions at more frequent intervals. This practice permits the treatment of more patients at less cost.

The excellent results obtained by the use of combined fever-chemotherapy in cases of asymptomatic neurosyphilis indicate that the patient's best interests are served by the prompt administration of this form of treatment.

It seems axiomatic to state that the best treatment for neurosyphilis is to detect it in its incipiency and to prevent the later development of its disastrous consequences by the prompt administration of some form of fever therapy combined with chemotherapy.

It is interesting to note that the remission rate of all of the 208 well-documented cases of various types of neurosyphilis was 52 per cent. An additional 28 per cent of patients were improved by the treatment. Thus, a total of 80 per cent of the patients were benefited.

Skin Diseases of the Ear. Reginald T. Brain.

Clin. J. **72**:49 (March-Apr.) 1943.

The field of physical therapy in dermatology is wide. Otologists are familiar with methods of ionization and are no doubt aware that although the penetration of ions is slight, the surface effect of the solutions employed is greatly intensified. This is an advantage in simple pus-coccal dermatitis, but eczematous lesions are usually aggravated by ionization. Although lesions

of the meatus can be treated with local ultraviolet light by using special quartz applicators and a Kromeyer lamp, the greater beneficial effect of the use of general ultraviolet therapy in chronic infections is often unsought. A drying source of heat is most comfortable and healing in the treatment of boils, and use should be made of infra-red rays, diathermy or roentgen rays. The latter in fractional doses is of the greatest service in many chronic skin diseases of the ear and it is unnecessary and less effective to attempt to restrict the irradiation to the meatal walls. Radium and radon are invaluable agents in the treatment of senile and actinic keratoses and epitheliomata.

Is the Hypersensitiveness to Chemical and Physical Agents Allergic in Nature? J. Bronfenbrenner.

J. Allergy **14**:105 (Jan.) 1943.

As to the mechanism responsible for release of histamine in heat and cold sensitivity, a plausible hypothesis has been offered by Grant, Pearson and Comeau which seems to harmonize with all the clinical observations. According to this hypothesis, the physical agents induce stimulation of efferent nerve fibers, releasing acetylcholine to the skin. This in turn releases histamine from the skin cells. In those persons who exhibit an abnormally intensified response to a normal release of acetylcholine at the nerve endings, symptoms of histamine poisoning of varying intensity result. Thus the abnormality is presumably related to the peculiar functioning of the cutaneous tissue not unlike that observed in cases of dermographia when an excess of histamine is released in response to mechanical stimuli. The apparent desensitization observed on repetition of the stimulus may be either of very short duration and presumably due to refractoriness of the vessels or it may be more lasting (forty-eight hours) if it is due to a failure of the skin to respond to acetylcholine and the resulting failure to liberate histamine.

It seems, therefore, that the term cold or heat allergy is not justified by the known facts and the term heat or cold intolerance should be substituted to avoid further confusion.

There are numerous reports that exposure to sunlight leads to urticaria, eczema, hives and prurigo and occasionally even formation of vesicles and bullae, referred to under the name of hydroya vacciniforme, also urticaria solaris. The most usual effect consists of erythema, edema and itching, followed later by pigmentation limited to the exposed area. If the reaction is intense, general symptoms not unlike sunstroke may be elicited and an intense local burn may result in necrosis. Thus, in general in photosensitive persons, reaction to visible light is identical with the reaction which normal persons exhibit on excessive exposure to short ultraviolet rays.

Whether the physical explanation of photosensitivity is the only mechanism involved is difficult to say, especially since on the one hand there are cases of light sensitivity without porphyria and, on the other, there are cases when porphyrin is present in large quantities without sensitivity to light and it has even been suggested that porphyrins may represent the result of skin injury by light rather than the cause of dermal sensitization. But in any event, it is unlikely that any real allergy (production of antibodies) to light exists. Although several authors claimed to have succeeded in transferring light sensitivity passively, the lack of proper controls minimizes the significance of such observations, especially in the light of findings to Callaway, who has recently detected the presence of a photosensitizing substance in the blood serum of a patient with abnormal sensitivity to light, which could readily account for the apparent success of some passive transfers.

Although efforts to find abnormal light absorption bands in the serum of photosensitive persons would represent a definite means of settling the problem of the nature of light sensitivity, such a procedure seems to have limited only promise due to the fact that tissues retain photosensitizing substances for a considerable period beyond the time when they can be found in the blood or urine.

The Action of Radiant-Heat Cradles. G. Malcolm Brown; David S. Evans, and K. Mendelssohn.

Brit. Med. J. **4280**:66 (Jan. 16) 1943.

This investigation was undertaken after two serious burns had occurred with the use of the heat cradle. Questioning of senior sisters revealed no standard procedure in the heat-cradling of cold or shocked patients and none was found in the literature. Aside from this lack of knowledge concerning the proper use of a powerful instrument, it was obvious that the cradle was needlessly dangerous and that the customary procedure which made it tolerable, the protection of the patient by a blanket, greatly reduced its efficiency. Heat administration should be as carefully controlled as the administration of drugs or oxygen.

By simple modifications a heat cradle has been devised which gives complete protection from early burns and which heats the patient much more quickly. The near infra-red component of the radiation has been converted with negligible energy loss to far infra-red, a type of radiation for which the skin has evidently a greater tolerance. This permits the withdrawal of the protection afforded by the blanket. The ability to control the dosage of heat accurately has resulted in the elimination of late burns and is of obvious value in careful treatment. With the shielded cradle it is possible to expose the patient in a heat bath at any temperature without ever exceeding that temperature.

There remain certain dangers. With the unshielded cradle the risk of burning the skin, or actual burns, must often have prevented the in-

troduction into the body of a dangerous amount of heat. When that costly safeguard is removed, more careful supervision of heat treatment is demanded. Under some conditions the patient's temperature rose at the rate of 2 F. in twenty minutes. This is a state which demands skilled observation and medical officers should supervise the use of the heat cradle more closely and not regard it as an instrument which can be left without instruction to the nursing staff.

The question of the place of heat in the treatment of shock is made more urgent by the more efficient cradle. There is little doubt that it can be overdone. Blalock and Mason have found that exsanguinated and traumatized animals survive longer if they are cooled rather than warmed and our own observations show that soon after the application of heat the blood pressure falls. Vasodilatation of the skin and enlargement of the vascular bed may well be harmful in shock. Some experimental work is being carried out to elucidate this point, but there is need for more observation on shocked patients.

Some instructions for the use of the cradle are: (1) Place the upper end of the cradle at the level of the lower end of the sternum. (2) Cover the cradle and its open ends with two blankets and do not disturb these unless absolutely necessary. (3) Leave the patient naked except for a small hand towel to protect the groins, and if the patient is restless insert the guard. (4) At first turn on all 12 bulbs. (5) After not more than thirty minutes reduce the bulbs to the number required to maintain the air temperature at the desired level. (6) Determine the oral temperature at least every fifteen minutes.

Physical Therapy of Peripheral Vascular Disease — Warshawsky

(Continued from page 494)

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		Entrance Requirements †	Length in Months	Classes Start	Tuition	Certificate, Degree, Diploma	Length in Months	Classes Start	Tuition	Certificate, Degree, Diploma
Children's Hospital, Los Angeles[1]	Steele F. Stewart, M.D.	a-b-c	6	FebAug	$200	Certificate	12	FebAug	$200	Diploma
College of Medical Evangelists, Los Angeles[1]	Fred B. Moor, M.D.	a-b-c	....	.....	.....	.....	12	JanJuly	$200	Certificate
University of California Hospital, San Francisco[1]	Frances Baker, M.D.	a-b-c	....	.....	.....	.....	12	Feb	$150	Certificate
Stanford University, Stanford University, Calif.[1]	Wm. H. Northway, M.D.	a-b-d[2]	7	Quart.	$286	Certificate	10	Quart.	$401	Cert. or Degree
Walter Reed General Hospital, Washington, D. C.	Capt. D. L. Rose, MC.	b	6	Quart.	None	Certificate	....	.....	.....	.....
Northwestern University Medical School, Chicago	John S. Coulter, M.D.	a-b-d	....	.....	.....	.....	9	JulyOct	$200	Certificate
State University of Iowa Medical School, Iowa City	W. D. Paul, M.D.	b-c	6	MarSept	None	Certificate	....	.....	.....	.....
Bouve-Boston School of Physical Education, Boston	Arthur L. Watkins, M.D.	HS	....Given in conjunction with Harvard				3-4 yrs.	Sept	$400 yr.	Dipl. or Degree
Harvard Medical School, Boston	Frank R. Ober, M.D.	a-b-c	6	June	$200	Certificate	9	June	$250	Certificate
Boston University Sargent College of Physical Education, Cambridge, Mass.	Louis Howard, M.D.	a-b-c	....	.....	.....	.....	24	JanOct	$435 yr.	Cert. & Degree
University of Minnesota, Minneapolis[1]	Miland E. Knapp, M.D.	a-b-c[3]	....	.....	.....	.....	12	Summer	$112[4]	Certificate
Mayo Clinic, Rochester, Minn.[1]	Frank H. Krusen, M.D.	a-b-c	6	Jan-July	None	Certificate	9	JanJuly	None	Certificate
Barnes Hospital, St. Louis	Frank H. Ewerhardt, M.D.	a-b-c	....	.....	.....	.....	9	Oct	$200	Certificate
St. Louis University School of Nursing, St. Louis[1]	Alexander J. Kotkis, M.D.	HS	....	.....	.....	.....	4 yrs.	JanSept	$250 yr.	Cert. or Degree
University of Buffalo School of Nursing, Buffalo[1]	George G. Martin, M.D.	a-b-c	6	FebSept	$420	Certificate	1-3 yrs.	FebSept	$350	Cert. or Degree
Hospital for Special Surgery, New York City[1]	Kristian G. Hansson, M.D.	a-b-c	....	.....	.....	.....	9	Sept	$300	Diploma
New York University, New York City[1]	William Bierman, M.D.	a-b-c	....	.....	.....	.....	9	FebSept	$396	Cert. & Degree
Cleveland Clinic Foundation Hospital, Cleveland	Walter J. Zeiter, M.D.	a-b-c	....	.....	.....	.....	9	Sept	None	Certificate
D. T. Watson School of Physiotherapy, Leetsdale, Pa.[1]	Jessie Wright, M.D.	a-b-c	....	.....	.....	.....	12	July	$200	Diploma
Graduate Hosp. of the Univ. of Pa., Philadelphia[1]	Wm. T. Johnson, M.D.	a-b-c	....	.....	.....	.....	12	Sept	$200	Certificate
Richmond Professional Institute, Richmond, Va.	T. W. Wheeldon, M.D.	a-d[5]	....	.....	.....	.....	9-12	Sept	$200-220	Certificate
University of Wisconsin Medical School, Madison[1]	Ernest A. Pohle, M.D.	a-b	6	FebSept	$96[4]	Certificate	12	Sept	$96[4]	Certificate

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MEETINGS OF PHYSICAL THERAPY ORGANIZATIONS

In these columns will be published information about meetings, election of officers, etc., of physical therapy organizations. New data should be sent promptly to the office of the Secretary, 2 E. 88th St., New York.

American Congress of Physical Therapy, 22nd Annual Session, Palmer House, Chicago, September 8, 9, 10, 11, 1943; **Instruction Course** to be held during the meeting; Dr. Richard Kovács, 2 East 88th Street, New York, Secretary. See announcements elsewhere this issue.

Connecticut Physical Therapy Society, meetings held in May and October at same time as the Connecticut State Medical Society. Dr. Karl Bretzfelder, 315 Whitney Avenue, New Haven, Conn., Secretary-Treasurer.

Southeastern and Southern Sections, American Congress of Physical Therapy will meet in conjunction with the *Section on Physical Therapy* of the *Southern Medical Association*, Cincinnati, Ohio, November 16, 17 and 18, 1943. Dr. E. J. C. Hildenbrand, 4201 Fessenden St., N. W., Washington, D. C., Chairman.

Kings County Medical Society, Physical Therapy Section; meetings at 1313 Bedford Avenue, Brooklyn, bi-monthly on second Thursdays; Dr. Samuel A. Warshaw, 1373 Ocean Parkway, Brooklyn, N. Y., Secretary.

The Penna. Academy of Physical Medicine; meetings at the Phila. County Medical Building, 21st and Spruce Streets, third Thursday, alternate months, starting with January; Dr. Harold Lefkoe, 5217 North Broadway, Philadelphia, Secretary-Treasurer.

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